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THE VENTRICULOMASTOID SHUNT.*†

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The patient who has increased intracranial pressure associated with obstructive or communicating hydrocephalus offers a distinct challenge from the surgical standpoint.

REVIEW OF LITERATURE.

Many procedures have been performed to shunt cerebrospinal fluid into various parts of the body. Davidoff,[1] in 1929, reviewed the treatment of hydrocephalus from ancient to modern times. Three years later, Davidoff and Bancroft[2] reported a case in which a ureterodural anastomosis had been performed for hydrocephalus. These authors credited Heile,[3] who wrote in 1925, as being the first to report on ureterodural anastomosis following unilateral nephrectomy for the treatment of hydrocephalus. Lehman,[4] in 1934, described his experiences with uretero-arachnoid anastomosis in three cases. Matson,[5] in 1949, presented a case report and description of arachnoid-ureterostomy via a polythene tube and,[6] in 1951, reported the successful relief of increased intracranial pressure in four children by ventriculo-ureterostomy.

Nosik,[7] in 1950, described the technique of ventriculomastoidectomy which he first performed on March 28, 1947. His technique consisted of shunting the cerebrospinal fluid

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from the lateral ventricle to the mastoid antrum. He made a cranial incision, inserted one end of a polythene tube into the ventricle and introduced the other end into the mastoid antrum through the petrous surface of the mastoid. In another article, also published in 1950,[8] he presented a case of marked hydrocephalus for which ventriculomastoidostomy had been performed three years previously.

Svien, Dodge, and Lake,[9] in 1952, described a modification of Nosik's operation. This also consisted of shunting the cerebrospinal fluid via a polythene tube, one end of which was inserted into the posterior horn of the lateral ventricle through a bur hole. They then brought the tube under the scalp to a second incision over the mastoid process and inserted the other end into the mastoid antrum after removal of enough mastoid cells to permit its entrance. These authors reported the case of a patient living 14 months after ventriculomastoid shunt had been performed for tumor of the midbrain with occlusion of the aqueduct of Sylvius. In the same year, Dodge, Miller, Lake, and Craig[10] reported a case in which this modification of Nosik's operation was used to relieve obstructive hydrocephalus. Their patient had undergone subtotal removal of an astrocytoma of the pons 14 years previously.

Matson,[11] in 1953, discussed results of arachnoid-ureterostomy in 50 children. Thirty-three of these children were living at the time of his report.

Brihaye and Périer,[12] in 1954, discussed the use of ventriculomastoid shunt and its complications in three of their cases. Ransohoff,[13] in 1954, described a ventriculopleural anastomosis which he used in six patients.

Hakim, Jiménez, and Rosas,[14] in 1955, described a technique of shunting cerebrospinal fluid from the spinal subarachnoid space to the spinal epidural space, and from a ventricle to the spinal epidural space. The result of each procedure on a patient was reviewed. Jackson and Snodgrass,[15] also in 1955, reviewed their experience with 62 patients studied over a four-year period after lumbar or ventricular peritoneal shunts had been performed.

Pudenz, Findlay, Hurd, and Shelden,[16] in 1957, described a

technique for shunting cerebrospinal fluid into the right auricle.

AIMS AND OBJECTIVES OF THIS STUDY.

No one of the many procedures developed to shunt the cerebrospinal fluid to various parts of the body has been consistently successful. For this reason and because many of the patients are desperately ill and need immediate relief, a simple procedure that can be done under local anesthesia, if necessary,

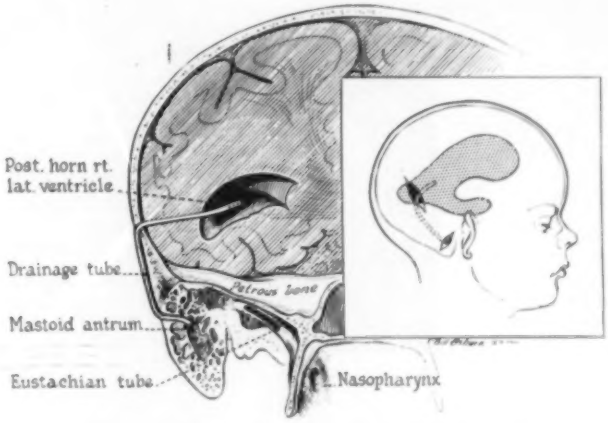

Fig. 1. Diagram of the ventriculomastoid shunt.

to relieve the increased intracranial pressure was sought. It is the purpose of this presentation to describe and discuss the ventriculomastoid shunt which was performed on 54 patients.

TECHNIQUE.

The modification of Nosik's operation as described by Svien, Dodge and Lake[9] with some further refinements was used on these 54 patients.

The procedure, as mentioned before, consists of shunting the cerebrospinal fluid from the lateral ventricle via a poly-

thene (polyethylene) tube, one end of which is placed in the lateral ventricle through a bur hole. The other end of the tube is passed under the scalp and inserted into the mastoid antrum, after the antrum is opened via a postauricular incision and enough of the mastoid air cells are removed to admit the tube into the antrum. A groove is made in the posterior edge of the incision in the bone to help hold the tube in place (see Fig. 1).

A further refinement in the technique was employed in these cases. It consists of removing the bulk of the mastoid

Fig. 2. Tantalum Button.

air cells and placing a tantalum button (see Fig. 2) in the defect in the mastoid cortex. The button is notched along the edge to admit and hold the polythene tube in place. The edges of the button may be trimmed to fit into the resultant defect in the bone. The soft tissues are sutured in place over the button after meticulous care in controlling all bleeding points. This refinement in technique has decreased the number of cases in which granulations have formed in the mastoid and subsequently have plugged the tube.

This modification of the ventriculomastoid shunt has the advantage of low risk and simplicity. It can be carried out under local anesthesia in the critically ill patient. Another advantage of this procedure is that the shunt can be explored

easily at either the ventricular or mastoid site if cerebrospinal fluid ceases to drain.

A simple test that will indicate a functioning ventriculomastoid shunt consists of turning the patient face down and observing cerebrospinal fluid escaping from the nose.

TABLE I.

Postoperative Follow-up and Results of Operation on 18 Patients with Congenital Hydrocephalus.

Ages at time of operation: 3 weeks to 3¼ years.

Case	Result
1.	Died 3 months and 3 weeks after operation.
2.	No follow-up.
3.	Alive 7 months after operation.
4.	Died 7 days after shunt; meningitis.
5.	Alive 11 months after operation.
6.	Alive 11 months after operation.
7.	Died 3 months after operation.
8.	No follow-up.
9.	Died 3 months after operation.
10.	Died 17½ months after operation.
11.	Alive 5 months after operation.
12.	Alive 6 months after operation.
13.	Died 4 months after operation.
14.	Died 8 months after operation.
15.	Alive 6 months after operation; head soft; doing well.
16.	Alive 12 months after operation; hydrocephalus had recurred.
17.	Died 7 months after operation; intracranial accident; shunt working.
18.	Died 2 months after operation.

Total living—7.

CLASSIFICATION OF CASES.

The 54 patients considered in this presentation were all those operated on from the initiation of the procedure in 1949 through the year 1955.

For study purposes, they have been divided into three groups: *1.* 18 patients with congenital hydrocephalus; *2.* 17 patients with increased intracranial pressure without brain tumor, and *3.* 19 patients with increased intracranial pressure with brain tumor.

Among the 18 patients with congenital hydrocephalus were six on whom a previous procedure had been done for relief of the hydrocephalus. Another patient had been operated on

for repair of spina bifida. Among the 17 patients with increased intracranial pressure without brain tumor, 14 had had previous operations for relief of the increased intracranial pressure. Among the 19 patients with increased intracranial pressure associated with brain tumor, 15 had had previous operations on the brain.

The results of the operations and the time of the follow-up are given in Tables I, II and III.

TABLE II.

Postoperative Follow-up and Results of Operation on 17 Patients with Increased Intracranial Pressure without Tumor.

Ages at time of operation: 17 months to 56 years.

Case	Result
19.	Died 3 months after operation; improved until death.
20.	Alive 2 years after operation; tube removed. Had meningitis before tube was removed.
21.	Alive and well 1½ years after operation; tube removed.
22.	Died 14 months after operation.
23.	Alive 4 years and 1 month after operation; 2 episodes of meningitis.
24.	Died 2 years and 1 month after operation.
25.	Alive 2 years and 1 month after operation; tube removed. Teaching school.
26.	Alive 7 months after operation; doing well.
27.	Died 1 month after operation.
28.	Died 2 months after operation.
29.	Died 2 months after operation.
30.	Alive 2½ years after operation; tube still in place.
31.	Died 1 year and 1 month after operation.
32.	Died 2 months after operation.
33.	Died 5 weeks after operation.
34.	Alive 2 years after operation; tube removed.
35.	Died 10 months after operation.

Total living—7; tube removed from 4.

In the congenital hydrocephalic group, four patients required revision of the shunt, and in one patient the other side was operated upon. Of the four revisions, two were done for obstruction at the mastoid end of the shunt by granulation tissue, and the other two were necessary because the tube had slipped out of the mastoid antrum.

In the group with increased intracranial pressure without tumor, three patients required revision of the shunt, and in one patient the tube was inserted on the other side. The

three revisions were done for obstruction from granulations in the mastoid end of the tube.

In the group with increased intracranial pressure associated with brain tumor, five patients required revision of their shunts. In one instance, the tube had slipped out of the mastoid antrum and, in the other instances, the tube was plugged with clots or granulation tissue.

TABLE III.

Postoperative Follow-up and Results of Operation on 19 Patients with Increased Intracranial Pressure with Intracranial Tumors.

Ages at time of operation: 2 years to 66 years.

Case	Result
36.	Died 10 days after operation.
37.	Died 2½ months after operation.
38.	Died 1 month and 3 weeks after operation.
39.	Died 2 months after operation.
40.	Died 3 months after operation.
41.	Alive 4 years after operation.
42.	Died 5 months after operation.
43.	Died 6 years after operation; another operation for brain tumor 2½ years after the shunt.
44.	Died 7 months after operation.
45.	Alive. Shunt failed after 2 weeks; then Torkildsen procedure and Roentgen therapy.
46.	Lived 2½ years; then died of meningitis. Had been rehabilitated.
47.	Died 1 month after operation.
48.	Died 6 months after operation.
49.	Died 2 months after operation.
50.	Died 8 months after operation.
51.	Died 2 months and 3 weeks after operation.
52.	Alive 1 year and 9 months after operation.
53.	Died 6 weeks after operation.
54.	Alive 6 years after operation.

Total living—3 after ventriculomastoid shunt; 1 after Torkildsen procedure and Roentgen therapy.

COMPLICATIONS.

Six patients with congenital hydrocephalus had meningitis postoperatively.

Among the patients with increased intracranial pressure without brain tumor, three patients had meningitis after operation; interestingly enough, this occurred over a year following the shunt in each instance. All recovered. In this group also were three patients who had infected cerebro-

spinal fluid at the time the shunt was established. Another patient in this group had bilateral pneumonitis after operation and recovered.

Of the patients with increased intracranial pressure associated with brain tumor, four had infected cerebrospinal fluid at the time the shunt was established. Two had meningitis after operation and recovered; one of these two had meningitis three times over a period of years. A third patient died of meningitis two-and-a-half years after operation. One patient in this group had pneumonitis and recovered.

SUMMARY AND CONCLUSIONS.

The literature pertaining to the shunting of cerebrospinal fluid to various parts of the body has been reviewed.

The technique of the ventriculomastoid shunt operation employed was a modification of the Nosik procedure. This operation was performed on 54 patients, on 18 for congenital hydrocephalus, on 17 for increased intracranial pressure without brain tumor, and on 19 for increased intracranial pressure with intracranial tumor.

From this study, the following comments may be made:

1. A simple procedure has been developed to shunt cerebrospinal fluid from a lateral ventricle to the mastoid antrum.

2. The procedure can, if necessary, be done on the desperately ill patient under local anesthesia.

3. In some instances, the operation has proved to be a life-saving procedure and, in a few instances, recovery has permitted removal of the tube.

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SIXTH INTERNATIONAL CONGRESS ON DISEASES OF THE CHEST.

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CHANGES IN THE VESTIBULAR LABYRINTH WITH INTENSE SOUND.*†‡

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INTRODUCTION.

During the course of studies on the effects of intense sound[1,2] on the cochlea of the guinea pig, it became apparent that the vestibular labyrinth was at times also histologically altered. The intensities and durations of the exposures that resulted in marked traumatic injury to the organ of Corti often produced some damage to the nonauditory labyrinth, although not of the extremely severe type described by Kimura and Perlman[3,4] for arterial and venous obstruction. The exposures to intense sound for one to four minutes for this series are of interest for comparison with the recent findings of McCabe and Lawrence[5] who reported collapse of the saccule in 58 per cent of their specimens subjected to a prolonged intense stimulus for 20 minutes. They utilized a random noise of 100 to 10,000 cps and permitted their animals to survive for 56 days, which presents more factors for comparison. Single frequencies were used in our experiments and the post-exposure lives varied from 0 to 133 days.

The selection of suitable material for a study of non-auditory injury proved to be a more arduous task than had previously been anticipated. In the beginning it was necessary to discard all specimens for which artifacts were in evidence in the

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vestibular labyrinth. This meant that a large number of preparations were made for the present study and only a few of these could be utilized. Rupture of thin membranes is not an uncommon finding in the celloidin embedded and sectioned temporal bones. This is true of Reissner's membrane in the cochlea and even more for the membranes of the utricle and saccule. An attempt was also made to select the material on the basis of frequency, duration and intensity of the exposures and post-exposure life. While we do not feel that all of these variables have been adequately covered, some results, nevertheless, afford an indication of correlation with histological changes. The results are, therefore, to be regarded as preliminary for which further experimentation may prove to be of value.

MATERIAL.

The basis of the present report is the findings for 46 serially sectioned and stained temporal bones of guinea pigs previously exposed to single frequencies varying from 170 to 50,000 cycles per second, and allowed to survive from 0 to 133 days. The traumatic stimulus for the 170 and 500 cps exposures was an enclosed acoustic system sealed to the right ear of each animal. The traumatic stimulus for the remainder of the frequencies was siren-generated, the only exception being the 13.2 kc. exposures for which a transducer employing the principle of the ribbon microphone was used. A description of the procedures and cochlear injuries for the latter two types of exposures has been previously reported (Covell and Eldredge[5]). The cochlear injuries for the enclosed system of exposures have likewise been described (Eldredge and Covell[6]).

Three different groups of animals were used as controls. One was comprised of temporal bone sections from six non-exposed guinea pigs, another was the right temporal bone sections of eight guinea pigs for which the ears had been exposed by the enclosed acoustic system to 500 cps at intensities varying from 118 to 133 db for five to 160 minutes, respectively, and a third group of seven sectioned temporal bones of five young dogs that were siren-exposed to 4, 11, and 18 kc. for

two to four minutes at intensities of 150 to 153.5 db. The temporal bone sections of the two ears of a non-exposed dog were also used for the study.

All animals were anesthetized with nembutal and perfused with physiological saline solution followed by Heidenhain-Susa fixative, following which the usual procedures for dehydration, embedding in celloidin, sectioning and staining with Harris hematoxylin and eosin were utilized.

FINDINGS.

The serial sections were examined for changes in the utricle and saccule and their maculas, the semicircular canals and their cristas and the nerve fibers related to these organs. Attention was given to a sheet of epithelial-like cells usually consisting of two to four layers which separated the utricle and the semicircular canals from the saccule and the remainder of the vestibule. It is considered to be a partition which divides the vestibule into inferior and superior portions. It was found to be particularly prominent in the guinea pig, by de Burlet,[7] who first described it in detail. It is similar in appearance to the membrana limitans (Waltner) that covers the scala tympani opening of the cochlear aqueduct. In the vestibule of the guinea pig's inner ear this partition is continuous with the lining of the vestibule, which is elsewhere a single layer of elongated cells with spindle-shaped nuclei. Since there is perilymph on each side of the membrane, its function must be primarily supportive for the utricle as it passes from one bony wall to the opposite side and is reflected onto the vestibular surface of the utricle. It also contributes support and attachment for the delicate connective cells in the superior part of the labyrinth. We have termed it "membranous partition" and it is referred to as such in this paper.

In order to simplify the interpretation of the results the material was divided into nine groups, according to the frequency of each exposure. Some of these groups include two different frequencies, which means that the number of specimens exposed to each frequency was too small. This was not done, however, when there were any significant differences in the vestibular findings relative to each frequency. The

post-exposure lives of all animals are not included. A rearrangement of the material according to the post-exposure life of each animal proved to disclose interesting findings which are considered later in the analysis of the results.

The observations do not include the ruptures of Reissner's membrane because there seemed to be no relation between this finding, which was verified in 17.3 per cent of all specimens, and ruptures in membranes of the vestibular labyrinth. On the other hand, marked injury to the organ of Corti, particularly in the basal turn, was more frequently accompanied by changes in the vestibular labyrinth than when marked injury occurred in the apical turns only and/or changes were of a mild nature in the basal turn.

Finally, there is brief mention of some degenerative changes in the maculas and detachments of otolithic membranes. Changes in the macula of the saccule occurred in approximately 12 per cent of the whole series while that of the utricle presented changes in only two specimens. It was felt, however, that they were mild and probably reversible, and that further studies with the use of electron microscopy would be necessary for a definitive evaluation of these degenerative processes. The otolithic membranes of both maculas were not consistently altered with any of the exposures. Loss of otoliths and detachment occurred in a few instances of the saccular macula but not for the utricular macula. The presence or absence of otoliths in these decalcified specimens as compared to similarly prepared unexposed controls was essentially the same.

The significant findings for each of the nine groups are given below.

Four animals were exposed to a frequency of 170 cps at an intensity of 146 db for one minute. There were marked changes in the organ of Corti in all but one of them, but no important changes occurred in the first turn of the cochlea. The vestibular changes in this group were very slight; there was a rupture of the membranous partition in only one specimen, and a slight collapse of the saccular membrane in another.

Four animals were exposed to 500 cps at an intensity of 142 db for one minute. Only one of them had marked lesions in the organ of Corti in the third, second and first turns while the remaining three had similar damage only in second and third turns. The membranous partition was found to be ruptured in three of the specimens, and all four had small ruptures in the saccular membrane, accompanied by collapse in two of them.

Three animals were exposed to 1 kc. at 150 db for one, two and four minutes, and five of their temporal bones were utilized in this study. Membranous partition ruptures were found in all five specimens, collapse of the saccular membrane was found in one and ruptures in two. The utricular membrane was ruptured in each labyrinth of one animal.

Five temporal bones from three animals exposed to 2 kc. at 142 db for one, two and four minutes, respectively, were found to present intense changes in the organ of Corti, primarily limited to the second turn of each cochlea. Membranous partition ruptures were found in four of the five specimens, and ruptures of the saccular membrane in three; one of them was accompanied by collapse. The utricular membrane was ruptured in four specimens.

Three animals were exposed to 4 kc. at 150, 153.5 and 163 db for two, two and one minutes, respectively. All of them had changes in the organ of Corti extending into the first turn, and in one of them there was a rupture of Reissner's membrane in the round window region. The membranous partition was ruptured in two of the three specimens and there was collapse of the saccule in one. The utricular membrane was collapsed in two specimens; one was also ruptured.

Two animals were exposed to the frequencies of 6 kc. and three to 10 kc. The intensities of the exposures were 142 and 153.5 db for 6 kc. and 156, 160.5, and 160.5 for 10 kc. The specimens subjected to 142 and 156 db were exposed for four and two minutes, respectively, while all others had one minute exposures. All specimens showed degenerative changes in the organ of Corti extending into the first turn, and in two of them into the round window region. The membranous

partition was ruptured in four of the five specimens. The saccular membrane was collapsed in one and ruptured in another. The utricular membrane was also ruptured in one specimen and collapsed in another.

Eight specimens were examined from five animals exposed to a frequency of 13.2 kc. at an intensity of 140 db for two

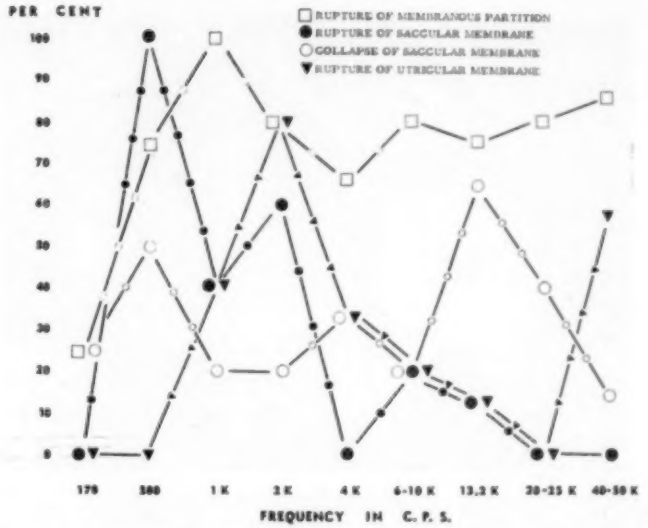

Fig. 1. In this chart are shown the percentage values for ruptures of membranes of the vestibular labyrinth as a function of frequency in 46 guinea pig ears exposed to intense sound. The results were grouped into nine categories of frequency exposures for reasons stated in the text. Clear squares—rupture of the membranous partition; solid circles—rupture of the saccular membrane; clear circles—collapse of the saccular membrane; solid triangles—rupture of the utricular membrane.

minutes. All of them had degenerative changes in the organ of Corti localized in the first turn, and these extended into the round window region in six specimens. Ruptures of the membranous partition were found in six of the specimens, and collapse of the saccular membrane in four; one of them was accompanied by rupture. In two of the collapsed saccules there were adhesions between the membrane and the macular

epithelium. The utricular membrane was collapsed in one specimen and ruptured in two others. One of the ruptures, however, had been repaired by connective tissue (see Fig. 5).

Five animals were exposed to the frequencies of 20 kc. (one specimen) and 25 kc. (four specimens), respectively, the intensities varied from 147 to 152 db for one to four minutes. With the exception of the specimen exposed to 20

Fig. 2. Photomicrograph of a section through the vestibular labyrinth of a guinea pig showing the relations between the saccule, utricle and lateral canal. This animal was exposed to 6 kc. at 142 db for 4 minutes and sacrificed eight days later. The only finding of any significance was a rupture of the membranous partition that is indicated by the arrow. LC—lateral canal; S—saccule; U—utricle; V—vestibule (X25).

kc., which had marked changes in the organ of Corti localized in the first turn, all others had intense degenerative lesions in the round window area. The membranous partition was ruptured in four specimens. The saccular membrane was intact in three, there being collapse in the other two, one of them with adhesion formation. In this group there were no changes in the utricular membrane. On the other hand one of the specimens showed the presence of leucocytes in the perilymphatic space of the posterior canal in the region of the ampulla.

Two animals exposed to 40 kc. at 140 and 144.5 db for two and four minutes respectively, and two animals exposed to 50 kc. at 142 db for four minutes furnished a total of seven temporal bones. The three specimens of the 40 kc. exposures presented marked changes in the organ of Corti localized at the round window region. Those exposed to 50 kc. did not present the same degree of degenerative alterations in the

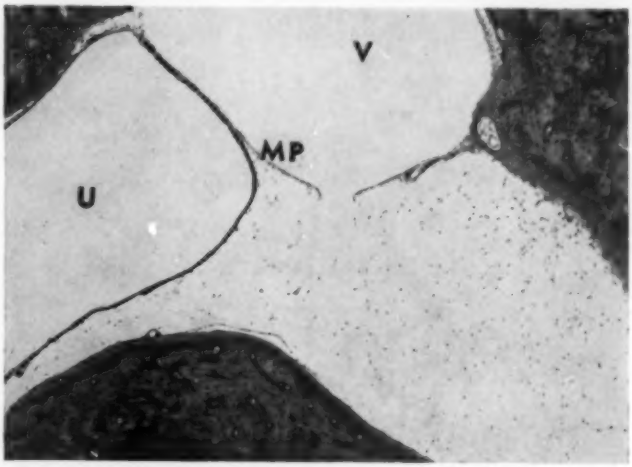

Fig. 3. Photomicrograph showing rupture of the membranous partition in the ear of a guinea pig that was exposed to 13.2 kc. at 140 db for two minutes and allowed to live for 133 days. The only other finding of significance was a collapse of the saccule, that was not unusual in animals with prolonged post exposure lives. MP—membranous partition; U—utricle; V—vestibule (X55).

organ of Corti. The membranous partition was found to be ruptured in six of the seven specimens, while the saccular membrane was intact in all specimens except one that showed collapse of the anterior end. Sloughing of the cells lining the vestibular side of the saccular membrane was found in four specimens, and some nuclei of the remaining cells were pycnotic. Displaced and pycnotic nuclei, as well as a deep eosinophilic cytoplasmic stain, were found in the cells of the macula of the saccule in all four specimens of the 50 kc. exposures and in one of the three exposed to 40 kc. The otolithic

membrane was detached, particularly at the anterior end, in three specimens and otoliths were found free in the saccule and attached to the saccular membrane. The utricular membrane was ruptured in four specimens, one of which presented two ruptures occurring in different places. Collapse was found in only one specimen. In three specimens hemorrhage and edema with leucocytic response were found in the tissue

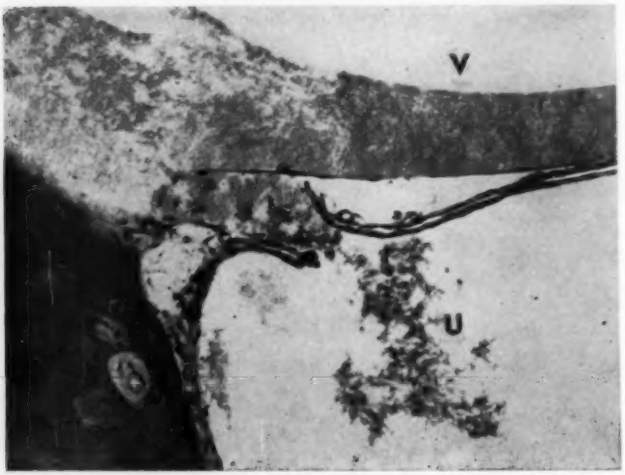

Fig. 4. Photomicrograph of a section through the ruptured portion of a utricular membrane in a guinea pig's ear that had been exposed to 2 kc. at 142 db for two minutes. The post exposure life was six days. This area in the inferior and medial wall of the utricle was found to be the commonest point of utricular rupture. U—utricle; V—vestibule (X220).

and around the nerve fibers from the macula of the utricle (see Fig. 7). The latter presented a normal appearance in all specimens except in one for which a few nuclei were pycnotic. The otolithic membrane of the macula of the utricle was without changes.

Fig. 1 summarizes the results obtained for each of the nine groups. The squares show the percentages of rupture of the membranous partition. Collapse of the saccular membrane is represented by clear circles, and solid circles represent

ruptures of the saccule. The triangles represent the ruptures of the utricular membrane. Collapse of the utricle is not shown because it is a less common finding and probably related to rupture of the membranous partition.

Rupture of the membranous partition (see Figs. 2, 3) varied from 70 to 100 per cent for all specimens exposed to

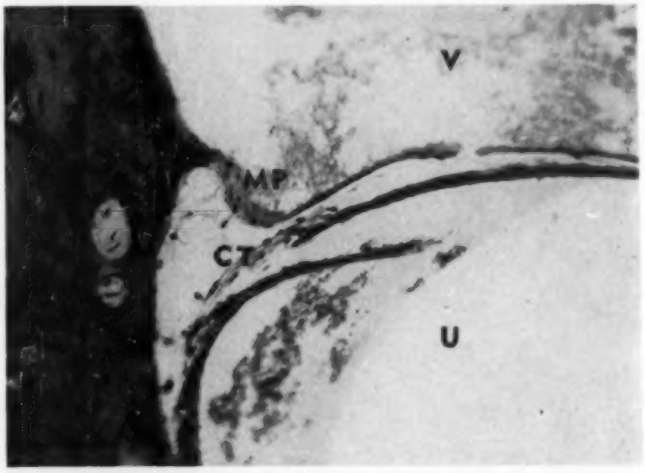

Fig. 5. Photomicrograph of a section through a ruptured utricular membrane of a guinea pig's ear exposed to 13.2 kc. at 140 db for two minutes. The animal was allowed to survive for 13½ days. There is an attempt on the part of the connective tissue to repair the rupture. The membranous partition is also ruptured and certain of its cells are in a degenerative state. CT—connective tissue; MP—membranous partition; U—utricle; V—vestibule (X220).

frequencies of 500 cps or above. Only 25 per cent of the 170 cps exposures revealed rupture of this partition.

Rupture of the saccular membrane occurred in all 500 cps exposures and became less with the increase in frequency. Collapse of the saccule (see Fig. 6) was not always accompanied by rupture and occurred chiefly for frequencies of 6 kc. or more, where it became more common than rupture.

Rupture of the utricular membrane (see Figs. 4, 5) occurred at frequencies of 1 kc. or above, and was present in 80 per

cent of the 2 kc. exposures and 30 and 40 per cent of the 4 kc. exposures, respectively. At higher frequencies of 6 to 25 kc. it was found in a small number of specimens, but again became significant for the 40 and 50 kc. group for which there was 57.1 per cent ruptures.

CONTROL GROUPS.

Serial sections through the vestibular labyrinths of a series

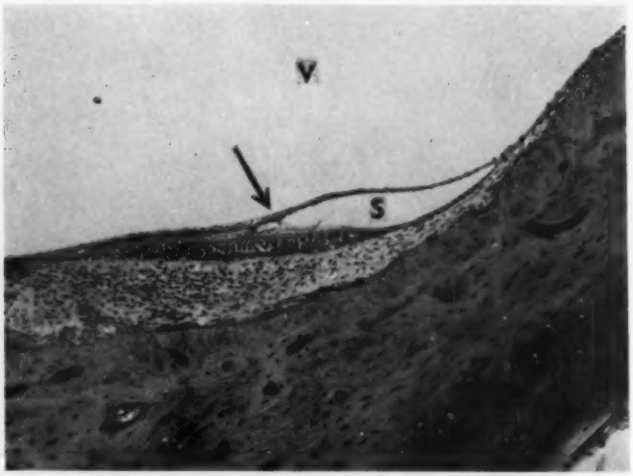

Fig. 6. Photomicrograph of a collapsed saccular membrane in the right ear of a guinea pig exposed to 500 cps at 140 db for one minute and sacrificed 14 days later. The membrane of the saccule was found to be ruptured at another level than illustrated. There is some tendency for adhesions (indicated by arrow) to form between the collapsed membrane and the cells of the macula. The latter reveal only slight degenerative changes, many of which are probably of a reversible type. S—saccule; V—vestibule (X115).

of temporal bones of six non-exposed guinea pigs served as controls. They were of the same source and ages as the exposed animals used for this study. The fixation, embedding, sectioning and staining were identical to the procedures used for the preparation of the temporal bones from guinea pigs exposed to intense sound. None of the changes described for the inner ears of the exposed guinea pigs appeared in these

specimens. These controls were particularly valuable in ascertaining some minor changes due to technical procedures such as occur in the otolithic membranes.

A group of eight guinea pigs, for which the right ears had been exposed to 500 cps was also used in a control study to compare the effects of lower intensities and longer durations of exposure. Four of these had been exposed at 118

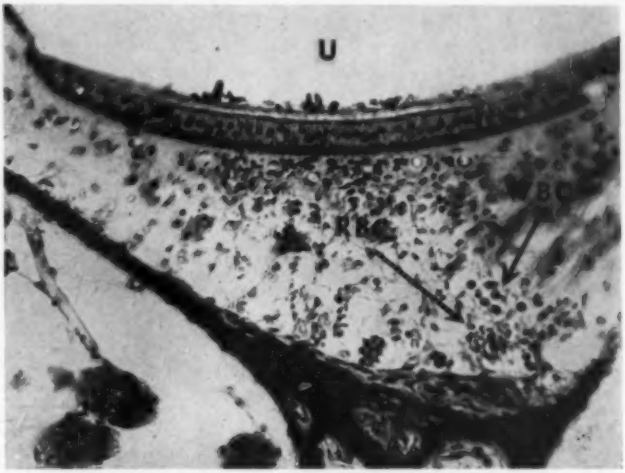

Fig. 7. Photomicrograph of a section through the macula of the utricle of a guinea pig's ear that was exposed to 50 kc. at 142 db for four minutes. The post exposure life was two days. There is evidence of hemorrhage and leucocytic response in the loose connective tissue and between the nerve fibers at the base of the macula. This specimen also revealed rupture of the membranous partition, collapse of the utricular membrane in its inferior portion, and sloughing of cells of the vestibular lining as well as those in the outermost layer of the saccular membrane. RBC—erythrocytes; U—utricle; WBC—leucocytes (X220).

db for 160 minutes; two were exposed at 128 db for 20 minutes, and two at 133 db for five minutes. All eight animals were sacrificed on the same day as the exposures. None of these except the latter two showed marked cochlear damage and rupture of Reissner's membrane. The latter was collapsed over the organ of Corti in several of the 160 minute exposures. The membranous partition of the vestibule was

found to be ruptured in six of the eight specimens, the saccular membrane was ruptured in one, but varying degrees of collapse of the saccule was apparent in five specimens. There was no rupture of the utricular membrane in any specimen, but there was evidence of partial collapse in four specimens.

Serial sections through seven temporal bones of five young siren exposed dogs[1,2] were examined for comparison of findings with those of the guinea pigs. Two of the dogs had been exposed to 4 kc. at 153.5 db for two and four minutes; one had been exposed to 11 kc. at 153.5 db for four minutes and two had been exposed to 18 kc. at 150 db for two and four minutes. The post-exposure lives varied from 0 to 13 days. There was severe injury to the organ of Corti in all specimens, with rupture of Reissner's membrane in four of the seven. The membranous partition of the vestibule was ruptured in five. The saccular membrane was ruptured in four specimens with the presence of a few red blood cells in the saccule in two specimens. There was no apparent collapse of the saccule in any of the specimens. The utricular membrane was ruptured in two and markedly collapsed in a third. Slight changes in the sensory and supporting cells of the macula of the saccule were in evidence in three specimens but none were apparent in the macula of the utricle. There were no apparent changes in the inner ears of the non-exposed dog used to control this study.

REVIEW OF FINDINGS.

The most common alteration in the vestibular labyrinth as a result of sound stimulus traumatic to the organ of Corti was found to be the rupture of the membranous partition that separates the vestibule into superior and inferior parts. It is doubtful that rupture of this membrane is of much, if any, functional significance unless it can result in extensive collapse of the utricle, a finding which was not encountered for any of the exposures. The inferior part of the utricle beneath the macula was in some specimens collapsed as a result of loss of the support of the membranous partition. The significance of such a finding is debatable. The percentage of the membranous partition ruptures showed no appreciable

changes that could be related with length of survival time of the animals. This agreed with the finding that there was no evidence of proliferation of the cells lining the vestibule in an attempt to repair these ruptures.

The next structure most commonly involved was the saccule. Rupture of the saccular membrane occurred in approximately one-fourth of the specimens, while collapse was found in one-third. There was a tendency for rupture to be more common to exposures of 500 to 2000 cps (inclusive) and for collapse to be more frequent than ruptures for exposures of 4 kc. and above. Rupture of the saccule did not appear to change with post-exposure life, which agreed with the lack of evidence of repair activity. Collapse, on the other hand, was much more common for the specimens with long lengths of survival time. From 0 to 2 days, 18.1 per cent of 11 animals showed collapse of the saccule, while 37.5 per cent of eight specimens showed collapse at 14 to 36 days. The percentage was increased to 71.4 per cent for seven specimens that were allowed to survive from 63 to 133 days.

Rupture of the utricular membrane occurred in approximately one-third of all specimens. The most frequent point of rupture was found to be below and medial to the macula in the vicinity of the utricular duct. It was more common for exposures of 1 to 4 kc. and for the 40 and 50 kc. exposures. There was a definite tendency for it to become less frequent with the increase in post-exposure life. This is probably due to repair of the rupture by connective tissue cells that proliferate from the connective tissue layer of the utricular membrane. Collapse of the utricular membrane was a less common finding, being present in approximately 15 per cent of the whole series. It did not show any relation to length of post-exposure life and was not found in specimens with intact membranous partitions.

The evidence for damage to the sensory and supporting cells of the macula was slight or even lacking in most preparations. The sensory cells for most of the 40 or 50 kc. exposures and for a few other specimens revealed displaced and pycnotic nuclei and the cytoplasm of the cells stained deeply with eosin. The inferior portion of the macula of the utricle showed

similar changes. Except for these there were no definite cytological changes in the utricular macula for any of the exposures. Loss of otoliths occurred in the saccular macula in several specimens, mainly in the 40 and 50 kc. exposures, and in some of these many otoliths appeared free in the saccule. Similar changes were not seen in the utricular macula.

Hemorrhage with leucocytic reaction and edema about the nerve fibers at the base of the macula of the utricle were found in two specimens exposed to 40 and 50 kc., respectively. Leucocytes appeared in the perilymphatic spaces of the semicircular canals and the utricle in two other specimens. One had been exposed to 13.2 kc. and the other to 25 kc.

In the specimens exposed to high frequencies of 40 and 50 kc. there was evidence of sloughing of cells of the vestibular surface of the saccular membrane, and some of the remaining nuclei were pycnotic. Similar findings were found for the utricular membrane although they were not so pronounced.

CONCLUSIONS.

1. Severe traumatic injury to the organ of Corti, caused by intense sound, particularly in the basal turn, is frequently accompanied by damage to membranes of the vestibular labyrinth.

2. The most common alteration in the vestibular labyrinth produced by instense sound is rupture of the "membranous partition."

3. The wall of the saccule was ruptured in approximately one-fourth of the specimens and collapsed in about one-third. Rupture was of highest incidence for exposures of 500 to 2000 cps and collapse was more in evidence for exposures of 4000 cps and above. The latter was also increased with prolonged post-exposure life.

4. The utricle was ruptured in approximately one-third and the inferior portion of it collapsed in about one-sixth of all specimens. Rupture was more common in 1 to 4 kc. and 40 and 50 kc. exposures. It was less frequent in specimens

with prolonged post-exposure lives, presumably because of the tendency for repair.

5. The sensory and supporting cells of the macula of the saccule revealed only a few changes of mild nature. Similar cells of the macula of the utricle were usually not altered.

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XII INTERNATIONAL CONGRESS OF LOGOPEDICS AND PHONIATRICS.

For the XII International Congress of Logopedics and Phoniatrics, Dr. Perelló has been in charge of the Rapport on "Functional Dysphonia," therefore, he requests every colleague to have something published about this matter and kindly send him a reprint for the record of said Rapport.

His address is: Dr. J. Perelló, Provenza, 319, Barcelona (9), Spain.

THE ROLE OF THE VOCAL CORDS IN HUMAN RESPIRATION.

Observations on Respiratory Function in the Human with Paralyzed Vocal Cords and Totally Absent Vocal Cords.*

JAMES J. O'NEIL,

Omaha, Nebr.

I. A REVIEW OF ALL PREVIOUS WORK AND LITERATURE ON THE PHYSIOLOGIC RESPIRATORY MECHANISM OF THE VOCAL CORDS. A PRESENTATION OF THE FOUR MAJOR CONCEPTS OF THE VOCAL CORDS IN RESPIRATION GAINED FROM THIS REVIEW.

The respiratory function of the larynx by the valvular action of the vocal cords has in the past been the subject of extensive investigative studies in animal and cadaveric experiments. Early studies by Longet,[19] Wyllie,[39] Brunton and Cash,[2] Semon,[34] Semon and Horsley,[36] were in agreement as to the valvular function of the cords of the larynx. Recent studies by Fink, Basek and Epanchin[11] and Feinstein,[8] employing photographic, roentgenologic, and electromyelographic techniques have offered additional confirmatory evidence of this function.

As far as we have been able to find, although Dionisio[6] reported dynamometric measurements for the pressure within the larynx of experimental animals, and Neumayer[25] reported manometric studies on the pressures within the human larynx, neither made graphic records. Hooper,[15] working with Professor Bowditch at Harvard, was one of the first to secure graphic records of the movements of the laryngeal cartilages on stimulation of the external branch of the superior laryngeal nerve. Murtaugh[21] obtained some excellent graphic records of abduction and adduction of the cords of the larynx

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of a cat and goat, and Feinstein,[8] employing electromyography, recorded changes in the human larynx. Fink, Basek, and Epanchin[10] obtained additional changes in the human by employing electromyography. In the information available, the graphic data obtained pertained only to changes within the larynx itself, and represented no particular influence of the larynx on respiratory ventilation.

After much consideration of a method that would provide graphic material for interpretation of the effect of the valvular cord action of the larynx on respiration, the utilization of accepted standardized pulmonary function tests seemed to afford an ideal laboratory method for conducting a scientific evaluation of the previously held concepts and theories on the effect of the vocal cords on respiratory ventilation.

To secure a completely accurate idea of previously and currently held concepts regarding the effect of this sphincteric valvular cordal effect on respiration, it was necessary to carry out a thorough review of the literature limited to this particular subject to provide the major concepts which could be subject to an accepted laboratory study, and to provide graphic material of the results for interpretation.

A great portion of this review revealed that the majority of previous investigative work had been confined to the larynx itself. For example, in 1841 Longet[19] reported his research on the function of the muscles and nerves of the larynx, emphasizing its valvular function. In 1866, Wyllie[37] reported similar findings in his observations on the physiology of the larynx. In 1882, Brunton and Cash[2] presented their study on the valvular action of the larynx, and in 1883, Hooper[15] showed the results of his experiments on the tension of the vocal bands. In 1890, he further reported his methods of demonstrating laryngeal movements. In the same year, Semon[35] reported on the position of the vocal cords in quiet respiration in man, and together with Horsley,[36] contributed a report on the experimental investigation of the central motor innervation of the larynx. In 1892, Dionisio[6] reported the dynamometric measurements of the pressure within the larynx of experimental animals, and in 1910, Neumayer[25] reported the pressures in the human larynx. Between 1902 and

1951, Onodi[27] established the principles of laryngeal innervation which were confirmed by Lahey,[17] Nordlund,[24] Ziegelman,[40] Lemere,[18] Terracol,[38] Furstenberg,[12] Murtaugh,[21] and Feinstein.[7] In 1929 and 1949, Negus[22,23] in his monumental studies on the larynx, reported the effects of the laryngeal vocal cords on respiration. In 1944, Pressman[29] reported further on the effects of the sphincteric action of the larynx on intra-abdominal pressure and on the muscular action of the pectoral girdle. In January, 1956, Fink, Basek and Epanchin[11] reported the laryngeal muscular activity in respiration by electromyography and X-ray studies.

It seems that the excellent work of the above investigators on the larynx resulted in an evolution of concepts regarding its effect on respiration. Negus[22,23] first stressed the importance of looking beyond the larynx in respiratory studies, for he feared that the very presence of the larynx may act as a hindrance by causing narrowing and obstruction to the air current. This search led to a study of the size of the glottis itself, the caliber of the tracheo-bronchial tree, the depth and rate of respiration, vital capcity, the size of the lungs, and the elimination of friction by the adaptability of the larynx.

Pressman,[29] in addition to making significant contributions on the effects of the sphincteric action of the larynx on intra-abdominal pressure and on muscular action of the pectoral girdle, has presented an excellent compendium of all the work of the previous investigators as it pertains to the role of the vocal cords on respiration.

The studies of Brunton and Cash,[2] Semon,[35] Negus,[22,23] Jackson,[16] and Pressman[29] postulate that the vocal cord action increases intra-thoracic and intra-abdominal pressure by trapping air within the tracheobronchial tree. In respiratory physiology this is important, primarily in circulatory changes and in cough. Negus[22,23] and Pressman[29] state that experimentally, when this valvular function is not functioning, there is about 20 per cent loss in ability to raise intra-abdominal pressure and a corresponding escape of air not under pressure.

Negus,[22,23] Jackson,[16] Rahn[30] and Riley,[32] in regard to the

role of the cords in producing a physiologic narrowing of the glottic chink, which supposedly effects an interchange of gases between the pulmonary alveoli and the blood stream, concur that oxygen passes through the alveoli walls into the blood stream, not during inspiration, but in expiration, with thoracic contraction, and that the rapidly increased intra-pulmonary pressure acting on the increased resistance of the partially adducted cords further increases the alveolar oxygen pressure and the speed of its passage into the blood stream.

Gordon,[13] Mills,[20] and Rahn,[31] in regard to vital capacity, agree that while it depends partly on the size and structure of the lungs, the rate of respiration is determined by the caliber of the air passages which cannot convey air at more than a certain speed. The ultimate efficiency of respiratory exchange should depend, therefore, upon the relationship of the glottic aperture to that of the trachea, as brought out by Negus[22,23] suggesting the tremendous importance of glottic action on respiratory physiology and its relationship to partial or complete opening and closing in the exclusion or retention of air. The movements of glottic dilatation at the beginning of inspiration and partial closure during expiration, both at times of exertion and during quiet respiration are thus explained.

As brought out by Bateman,[1] Fowler,[11] Pressman,[29] and Rahn,[30] in man, with the finely divided air spaces and large volumes of supplemental and residual air, there is actually no need for any immediate retention of air by glottic action for effective function. If the glottis is partially closed at the beginning of expiration, bronchial musculature may force some of the air backward into the distant alveoli, assisting the process of diffusion. If obstructed on inspiration, bronchial dilatation will abstract air from the alveoli to mix with the in-rushing uncontaminated tidal air.

Negus[22,23] maintains a regulatory function for the cords in ventilation. By its obstructive action the glottis can retard the filling or emptying of the bronchi, and thus delay the Herring-Breuer reflex, which provides time for the adequate exchange of gasses in the lungs. This keeps the O_2 and CO_2 contents of the blood at the right level. It is the complete

dilatation of the bronchi at the end of inspiration which sends a reflex stimulus to the respiratory center through the afferent vagal fibers; this stimulus initiates expiration. The reverse effect occurs at the end of expiration, when the bronchi are contracted.

Numerous investigators, including Dean,[5] Fenn,[8] Haldane,[14] Otis,[28] and Pressman,[29] have felt that glottic obstruction to the entry of air during inspiration adds to the reduction of intra-pulmonary and intra-thoracic pressure. This produces pressure on the great veins and the right auricle, and sucks blood back into the heart during inspiration. Descent of the diaphragm at the same time squeezes blood from the splanchnic area into the vena cava, and thus to the right auricle. It causes dilatation of the pulmonary capillaries and facilitates the flow of blood through the lungs, thus providing a large volume of blood deficient in oxygen and overcharged with CO_2 to meet the newly entering tidal air which fills the dilating alveoli. The rush of blood through the lung delivers a copious supply to the left side of the heart, and according to the law of the heart, the left ventricle, therefore, contracts more forcefully and raises the aortic and general arterial pressure; in expiration, the reverse is true. Obstruction throws back pressure on the venous circulation, slowing it at a time when the emptying of the lungs does not allow for active respiratory exchanges. The pulmonary capillaries are compressed in unison, with the partial collapse of the alveoli. There is, however, a temporary rise of aortic pressure at the beginning of expiration, since compression of the lungs by the expiratory effort squeezes blood into the left side of the heart.

Negus[22,23] has ascribed two additional functions to the vocal cords in respiration: *1.* the main regulating valve supplying the fine adjustments in augmenting the obstructive action, correlating the volume of blood and its rate of passage through the lungs with the volume of air in the alveoli, especially to its contents of oxygen and carbon dioxide; *2.* adapting the size of the lumen of the airway to the needs of the moment.

The vocal cords represent a point of constriction in the lower respiratory tract which is variable in degree, depending

upon the position of abduction or adduction assumed by the cords. In inspiration, as in singing, Pressman[29] has shown that the cords are so abducted they can scarcely be distinguished from the lateral walls of the larynx; in expiration, there is marked narrowing of the glottic chink. Pressman concludes, therefore, that in each respiratory cycle there is a to-and-fro movement which is naturally more conspicuous with deep breathing than with shallow breathing at rest. The marked degree of approximation on expiration establishes a point of relative resistance to the escape of air with a momentary increase of intra-tracheal, intra-bronchial, and intra-alveolar pressures. This elevated pressure increases the oxygen flow from the alveoli through the alveolar walls into the blood stream. The vocal cord action rapidly increases the pressure to insure rapid absorption of large quantities of oxygen. In addition to these purposeful movements of the vocal cords, there is a lowering, backward displacement and lateral deviation to the left of the entire larynx. These movements are due to intra-thoracic pressure variations, are of small amplitude, and are not universally present.

This review of all previous work and literature reveals that four major respiratory functions of the vocal cords have been ascribed:

1. Regulation of gaseous transport across the alveolar membrane by increasing intra-thoracic pressure during expiration.

2. Regulation of alveolar filling by forced passage of gas into unaerated segments of the lungs on expiration.

3. Regulation of cardiac output by effecting a rise in pulmonary pressure with decreasing venous return during expiration.

4. Control of the mass motion of gas during ventilation with the cords imposing a time element to expiration.

II. RE-STATEMENT, ANALYSIS, AND DISCUSSION OF FOUR CONCEPTS OF VOCAL CORD ACTION IN RESPIRATION GAINED FROM REVIEW OF LITERATURE.

Statement of Aims and Objectives of Present Study. Methods

Employed in Study. Brief Clinical Resume of Subjects Used. Brief Description of Tests Used. Discussion of Results.

From this historical review it is apparent that the role of the vocal cords in respiratory function has been long accepted. A number of theories have been advanced, each describing various mechanisms by which the vocal cords control or regulate some phase of respiration; however, experimental work to support these findings has for the most part been confined to studies of the larynx itself, and is inadequate. Much of this experimental data has been obtained from lower animals, and is of questionable validity when applied to the human. Although a wealth of data dealing with respiratory function has been accumulated in recent years, little or no attempt has been made to analyze the role of the vocal cords in the complex mechanism of respiration, so that the accepted theories have remained unquestioned. Recent interest and activity in surgery of the larynx, trachea, and thyroid have prompted intensive anatomic, neurological, roentgenological, and electromyographic studies of the cords in phonation; however, their time honored role in respiration has been unchallenged, obscure and explainable only by primitive concepts.

It is apparent that all of the four major respiratory functions of the vocal cords revealed by a review of literature are fundamentally similar, in that they place the vocal cords as the regulator of respiration in the expiratory phase, and that each theory deals with the effects of elevated expiratory pressure on some phase of ventilation or gas exchange.

The first theory, *i.e.*, the regulation of gaseous transport across the alveolar membrane by increasing intra-thoracic pressure during expiration, was first proposed by Negus,[22,23] and supposes that with cord adduction in expiration the rise in intra-pulmonary pressure effectively elevates the partial pressure of oxygen and thus increases oxygen transport across the alveolar membrane. Although this concept of expiratory predominance in gas exchange seems logical and actually occurs in some lower vertebrates, it is not quantitively significant in the human, as reference to the Oxygen Dissociation Curve discloses.[4]

The second theory, *i.e.*, the regulation of alveolar filling by forced passage of gas into unaerated segments of the lung during expiration, states that the rise in expiratory pressure effected by the cords forces inspired gas into alveoli non-aerated. This concept is no longer tenable in light of the recent work of Roos, Murphy, and Dahlstrom[23] that ventilation is not enhanced by increase of intra-pulmonary pressure.

The third theory, *i.e.*, the regulation of cardiac output, by effecting a rise in pulmonary pressure with decreasing venous return, supposes that the diminshed venous return attendant to expiration is the result of cord adduction with a rise in pulmonary pressure. While this is true in the classic Valsalva maneuver, it is not the case in basal respiration, where the increase in intra-pulmonary pressure is directly related to the elastic recoil of the lung which occurs with the end of inspiration.

The final theory, *i.e.*, the control of the mass motion of gas during ventilation with the cords imposing a time element to expiration, concerns the role of the vocal cords in minute to minute regulation of expiration, and its effect on expiration time rather than pressure. Although this theory has no experimental background, it is in keeping with modern physiologic concepts of the effect of time as an adjunct to intra-pulmonary mixing with the end-result the creation of homogenous substrate to promote even trans-alveolar exchange of oxygen and carbon dioxide.

In view of the sparse or inadequate experimental work to support any of these theories in human respiration, it seemed logical to reevaluate the role of the vocal cords in respiration. To this end the application of the modern respiratory function tests appeared admirably suited. It was the aim and objective of this study to take two groups of subjects representing the extremes in vocal cord abnormalities, and subject them to the entire battery of respiratory function studies, including ventilation and mixing analysis.

The first group of subjects studied had various types of unilateral and bilateral cord paralysis presenting marked narrowing of the laryngeal aperture. In this group were two

women and three men in good general health, with normal chest X-rays, and laryngoscopic evidence of vocal cord paralysis. In one woman bilateral cord paralysis followed a thyroidectomy. The other woman's unilateral cord paralysis was spontaneous for which careful laryngological, medical, and neurological work-up failed to determine the etiology. The three men had unilateral cord paralysis following radical neck dissection for carcinoma.

The second group studied were subjects without vocal cords as the result of total laryngectomy for carcinoma of the larynx. They were all white males in good general health with normal chest X-rays and were free of any evidence of carcinoma.

The subjects of both Group I and Group II were free of any evidence of unrelated cardio-respiratory disease or subjective signs of respiratory deficiency.

While the various techniques for evaluating pulmonary function have individual limitations, when combined, these tests offer an accurate index of the subject's ability to move adequate volumes of gas in and out of the lungs within a specified time, and an evaluation of the intra-pulmonary mixing of the inspired and residual gases of the lung.

The test battery utilized in this study was arranged into two groups: the first comprised the following pulmonary function tests which were classic for evaluating accurately ventilatory mechanics with an assay of respiration in all phases, time, and maximum effort:

Tidal volume, defined as the volume of gas the patient is breathing quietly and at rest.

Minute volume, defined as the volume of air entering and leaving the lungs per breath per minute.

Functional residual capacity, defined as the volume of gas remaining in the lungs after a natural expiration.

Vital capacity, defined as the volume of gas that can be expelled by a maximal expiration following a maximal inspiration.

Exploratory reserve volume, defined as the volume of gas remaining in the lungs after a maximal expiration.

Maximal breathing capacity, defined as the maximal volume of gas that can be breathed per unit time.

End tidal CO_2 determination, defined as the pressure in mm. of CO_2 gas in last portion of expiration.

Inspiratory capacity, defined as the maximal volume of air that can be inspired from the end of a normal expiration.

Timed vital capacity, defined as the performance of vital capacity in relation to time.

Total lung capacity, defined as the sum of vital capacity and residual capacity.

Residual volume, defined as the volume of gas remaining in the lungs after a maximal expiration.

The second group of pulmonary function studies were used to analyze the functional effects of ventilation, including the evenness of gaseous mixing and the elasticity of the tracheobronchial tree and the parenchyma:

Fowler's single breath test, defined as the method of determining the evenness of ventilation.

Nitrogen equilibration, defined as the method of determining the functional residual volume of the respiratory system. The time required is analagous to the seven-minute Cournand Washout Test, and measures the evenness of ventilation when the dead space and ventilation pattern are normal.

The elastance and resistance were studied by recording the maximal expiratory and inspiratory pressures by sinusoidal wave, by outflow curves, and by maximal flow velocities.

All testing was carried out under basal conditions with the norms for age, height, and weight drawn from the standard table of Cournand and Richard, Comroe, et al.[4]

The results of this study show gross abnormalities in some phase of testing in both groups. In Group I, all subjects had low maximal breathing capacities with the exception of

one, who was borderline normal. In Group II, all subjects had low maximal breathing capacities and additionally had abnormalities in mixing, as shown in the Fowler single breath tests; there was one possible exception to this.

In Group I the deficit in the maximal breathing capacity was observed to accompany a rise in the functional residual capacity to the upper limits of normal. These findings are similar to those patients studied elsewhere with clinical emphysema of the acquired or senile type. It seems apparent that the expiratory obstruction attendant to cord paralysis results in a loss of timed breathing capacity with eventual loss of pulmonary elastance and rise in residual volumes. The one exception to this is the subject who was the borderline limit of normal.

In Group II all subjects had lowered maximal breathing capacities, and with one exception impaired intra-pulmonary mixing as shown in the Fowler single breath tests. It is of special interest that the loss of pulmonary elastance is not a component here as in Group I, but rather demonstrates the deficit in effective time and mass gas motion and mixing attendant to the absence of vocal cords.

SUMMARY AND CONCLUSIONS.

We have presented a review of all previous work and literature on the physiologic mechanism of the sphincteric vocal cord action in respiration. From the historical review, four major concepts of cord function were presented, summarized and discussed. In the absence of adequate experimental work to support these theories in the human, and with newer methods of accepted respiratory study now available, it was felt that re-evaluation of these theories would be in order. To accomplish this, two groups of subjects representing the extremes in vocal cord abnormality were subjected to the entire battery of respiratory function studies. To our knowledge this study represents the initial application of the pulmonary function tests to an evaluation of vocal cord action in human respiration.

From our review of the literature on the sphincteric action

of the vocal cords on respiration, and a careful study of the results of pulmonary function tests carried out in subjects with the extremes in vocal cord abnormalities, we can conclude:

1. That the gaseous transport across the alveolar membrane by an increase of intra-thoracic pressure produced by the cords in expiration is quantitatively insignificant in the human.

2. That ventilation in the human is not enhanced by an increase of intra-pulmonary pressure produced by the cords in expiration.

3. That cardiac output is not affected by cord adduction in expiration with the basal respiratory conditions prevailing in the human where the increase in intra-pulmonary pressure is directly related to the elastic recoil of the lung which occurs with the end of inspiration.

4. That laryngeal paralysis in the human results in a definite decrease in maximal breathing capacity in the majority of subjects studied with a rise in the functional residual capacity to the upper limits of normal.

5. That laryngeal paralysis in the human produces a loss of breathing capacity (timed) with eventual loss of pulmonary elasticity and rise in residual volumes.

6. That in the majority of subjects studied, total vocal cord absence in the human results in lowered maximal breathing capacities and impaired intra-pulmonary mixing.

7. That total vocal cord absence has less effect on pulmonary elastance than cord paralysis.

8. That total cord absence creates a significant deficit in time and mass gas motion and mixing.

9. That both cord paralysis and total cord absence is not a physiological innocuous occurrence, even in subjects clinically free of symptoms of respiratory deficit.

10. Finally, that the absence of normal vocal cord function,

either from paralysis or laryngectomy, results in a definite measurable physiologic deficit in respiratory function.

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612 Medical Arts Bldg.

CHRONIC ENLARGEMENT OF THE PAROTID GLAND; HYPERTROPHY AND FATTY INFILTRATION.*

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The occurrence of chronic bilateral non-inflammatory parotid swelling is well recognized in many malnourished peoples and in patients with chronic alcoholism, obesity and portal cirrhosis.

Long standing bilateral asymptomatic parotid swelling has been described in the following undernourished groups: Egyptian peasants, certain hill people of Madagascar, Bantus in Africa, some Negro groups in Brazil and in parts of Malaya and rural India. Although the condition has been seen associated with pellagra, the general opinion is that a diet which is generally inadequate, rather than a specific deficiency is involved. Bilateral parotid swelling is also seen in patients recovering from severe malnutrition, such as infants recovering from kwashiorkor disease, and in emaciated prisoners repatriated from Russian prisoner of war camps. Histopathological observations on the parotid are relatively few, but generally show a hypertrophy of the secreting cells with increased number of zymogen granules and a striking absence of an inflammatory reaction. Sialograms when done have been reported as normal.

The literature on this subject has been extensively reviewed by Sandstead, et al.,[1] in 1955. They report nutritional and clinical studies on three groups: an Asian population, 1094 mental patients in Saint Elizabeth's Hospital, D. C., and 165 patients in a hospital for American Indians in the Southwest. They found parotid enlargement in 11.6 per cent of the Asians, 3.5 per cent of the mental patients, and in 12 per cent of the

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Indians. The swelling was bilateral and symmetrical and was occasionally accompanied by an increase in size of the submaxillary glands. Parotid function, as estimated by the thiocyanote content of the saliva was normal. No evidence of virus infection was found on growing tissue cultures from five biopsy specimens. Biopsies were obtained from eight of the enlarged parotids. No inflammatory or obstructive changes were seen to explain the enlargement. The St. Elizabeth's hospital group showed abundant intralobular fat but normal cells, acini and ducts. It was thought possible that the fat might be responsible for the enlargement, but it was not present in amounts greater than in normal parotid glands. Microscopic examination of the Asian cases showed enlarged parotid lobules with minimal intralobular fat. The enlargement seemed to be due to enlargement of the individual cells, which are seen to be packed with zymogen granules. They conclude, "The enlargement in the acute phase appeared to be due to swelling of the individual cells and in the chronic phase to a replacement of acinar tissue with fat." After extensive nutritional surveys on these patients they conclude that bilateral chronic asymptomatic swelling of the parotid gland in these cases is a manifestation of recent or remote malnutrition.

Dechame. et al.,[2] published a study on a series of French patients with bilateral parotid enlargement which they termed "essential." They classify their cases into four groups on the basis of sialograms and the interpretation of needle biopsies. Their first two groups appear to show hypertrophy, while the last two seem to be definitely inflammatory. Du Plessis,[3] of Capetown, South Africa, states that parotid enlargement in chronically malnourished people or those recovering from severe nutritional deficiencies is due to an increase in the size of the acini. The individual cells are distended with secretory granules and are enlarged up to 50 per cent of normal. The glands will revert to normal if adequate nourishment is provided. Salamon[4] describes parotid swelling in nine cases of pellagra. Five biopsies were done. The specimens showed no pathological changes. All showed numerous secretory granules in the acinar cells, suggesting parotid hyperfunction.

Takaoka, et al.,[5] from experimental work suggest that parotid enlargement may be associated with disturbance in the serum proteins. John[6] reports bilateral parotid enlargement in some cases of diabetes mellitus; however, no pathological findings are described.

Enlarged parotid glands have also been reported in patients with alcoholic cirrhosis and in obese alcoholics without definite evidence of cirrhosis.

Sposito and Chili[7] obtained parotid tissue from six cases of portal cirrhosis. Using a 2 mm. needle, he removed a core of gland through a small skin incision. All cases showed glandular hyperplasia with moderate increase in fatty tissue.

Bonnin, et al.,[8] and Bonnin,[9] from Bordeaux, France, report bilateral parotid swelling in 80 per cent of patients with cirrhosis. Pathological findings in a few cases showed hypertrophy of the acinar cells with complete absence of inflammatory changes. They report an "aquiparus" appearance of the secreting cells, which seems to mean a pale vacuolated cytoplasm with few or no zymogen granules resembling a mucous rather than a serous gland. This appearance may be due to fatigue of the cell after prolonged and excessive stimulation. These authors state that this is the characteristic appearance in the parotids of cirrhosis. Fat was found in normal proportion.

Rothbell and Duggan,[10] of Syracuse, New York, describe six obese patients with liver disease in whom parotid enlargement was a striking finding. Parotid biopsy was done on four cases and revealed various degrees of fatty infiltration. They conclude that the parotid enlargement seems to have much the same significance as a fatty liver, pointing to disturbed nutrition but not defining the disturbance. Wolfe, et al.,[11] of Boston, present clinical and laboratory findings on 16 patients with hepatic cirrhosis associated with chronic alcoholism who had bilateral symmetrical parotid swelling. In some cases, improvement in liver function was accompanied by reduction in the size of the parotid glands. No pathological findings in the parotid were described, but from a study of the literature, they conclude there was no essential histologic difference

between the parotid glands of malnourished persons and those of alcoholic patients. They conclude that the pathogenesis of the parotid enlargement in liver disease is unexplained, but is probably related to similar changes reported in malnourished persons.

Duggan and Rothbell[12] studied 50 persons with non-inflammatory parotid enlargement. On the basis of dietary history the patients fit broadly into three categories: *1.* Abnormal with alcoholism—41 cases; *2.* abnormal without alcoholism (3 cases), and unremarkable (6 cases). The parotids were both enlarged but not always asymmetrically. Enlargement was diffuse and asymptomatic. Obesity and sthenic habitus was common. Thirty-five patients had diastolic hypertension, and hepatomegaly was found in 37. No pathological data is presented here. After a review of the subject they state that non-inflammatory parotid enlargement is without morphological definition, of unknown cause and of unknown pathogenesis; however, it always seems to be associated with disturbed nutrition, possibly a protein deficiency. Enlarged parotids are strongly suggestive of associated liver disease, and provide an indication for the study of glucose tolerance.

Fatty infiltration of the parotid has been described occurring in the absence of dietary disturbances. In these instances, the change can be unilateral or bilateral, and diffuse or localized.

Simonetta[13] describes six cases of a condition which he names Lipomatous Pseudohypertrophy of the Parotid Gland: four were unilateral and two were bilateral. Two cases were biopsied and revealed diffuse fatty degeneration of the entire gland. The proportion of fat cells to acini varied in different portions of the gland, but there was always more than 50 per cent fat. The secretory elements were normal. No cause for the disturbance was found.

Gilman, Schwartz and Gilman[14] report clinical and pathological findings in a thin female American Indian with a discrete mass in the left parotid which was clinically indistinguishable from a benign neoplasm. On surgical removal of the mass the histological picture was "parotid gland with

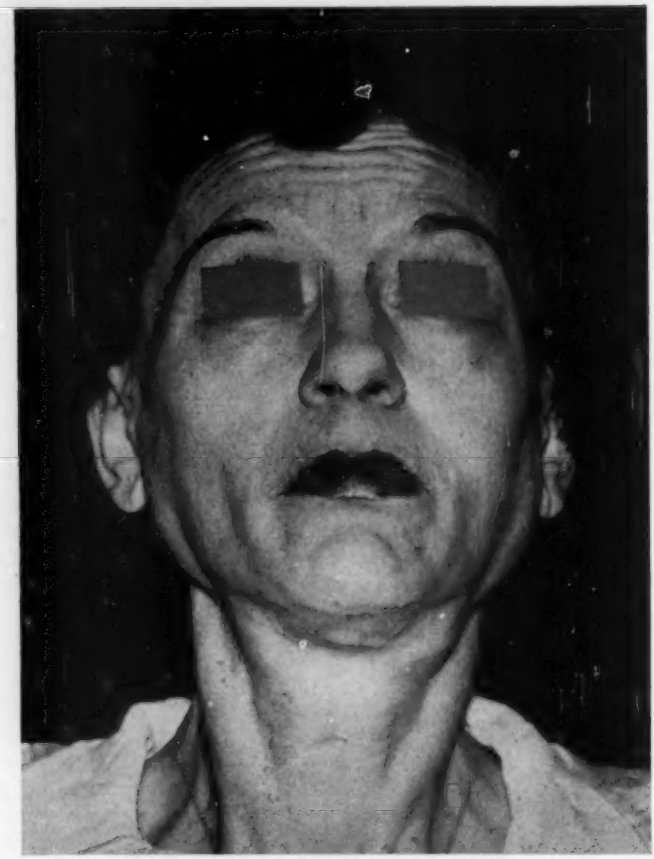

Fig. 1. Case 1. A 39-year-old woman with chronic salivary gland swelling for 18 months.

a marked fatty infiltration separating the individual acini into groups of various sizes."

For several years the authors have biopsied all diffusely enlarged parotid glands on which the diagnosis was in doubt. Biopsy of the parotid gland is an easily accomplished and harmless procedure. Under local anesthesia, a small skin

incision is made over the parotid below the ear canal and a portion of the superficial lobe of the parotid is removed. There is no danger to the facial nerve, and a fistula will not develop. If the patient is concerned about the small scar, it can be made more inconspicuous by separating the ear lobule from the face.

Following are brief case reports of seven patients who pre-

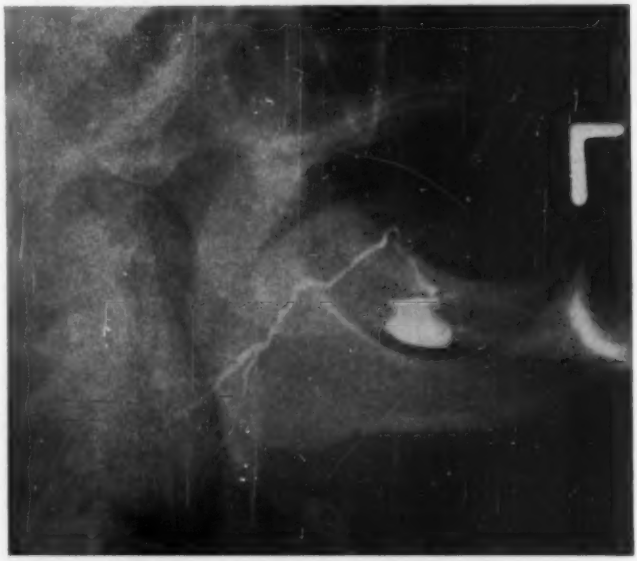

Fig. 2-a. Case 1. Normal parotid sialogram.

sented with parotid enlargements which were neither inflammatory nor neoplastic.

Case 1. 726468. A 39-year-old white female (see Fig. 1). Asymptomatic enlargement of both parotid glands, and the left submaxillary gland had been noted for over 18 months. The submaxillary gland had been biopsied elsewhere, and the patient was assured that the condition was benign. When seen in consultation, she was a patient on a medical service being studied for generalized malnutrition and pulmonary tuberculosis. She weighed only 80 pounds, 8 ounces. There was no evidence of alcoholism. In addition, she showed evidence of a severe psychoneurosis. Left parotid (see Fig. 2-a) and left submaxillary sialograms

were within normal limits. A biopsy of the left parotid was done (see Fig. 2-b). The acinar cells were swollen and stuffed with zymogen granules. The acini were almost double the normal size. The ducts were normal. Practically no fat cells were present. A review of the submaxillary biopsy revealed a similar picture. At present the patient is under treatment for pulmonary tuberculosis and an effort is being made to improve her nutritional status in the hope that the salivary gland swelling will recede.

Case 2. J.B., 701568. A 41-year-old white female. Bilateral diffuse parotid enlargement had been present for at least two years. Biopsy

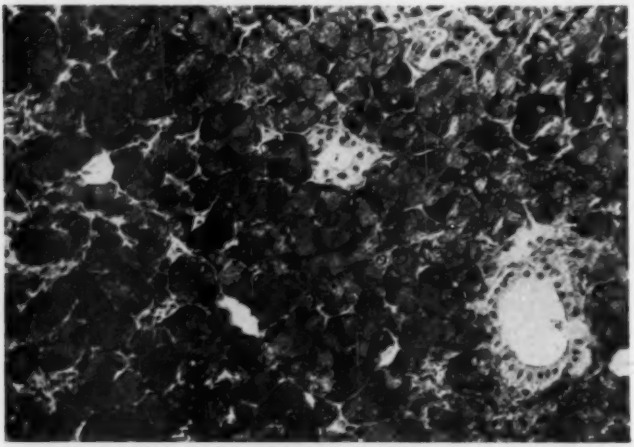

Fig. 2-b. Case 1. Left parotid biopsy. There is no inflammatory reaction, but a striking increase in the number of zymogen granules. The acini are almost double the normal size.

revealed ideopathic hypertrophy with a small amount of fat. Although she had many complaints, a thorough medical work-up revealed no evidence of systemic disease. There was no obvious nutritional disturbance or excess intake of alcohol.

Case 3. S.G., 663246. A 46-year-old white female (see Fig. 3-a). Bilateral painless parotid enlargement had been present for many years. She was a very obese sthenic person who consumed several ounces of wine daily. Three X-ray treatments had been given to each parotid area without improvement. She did not complain of dry mouth. Liver function tests were within normal limits, but a clinical diagnosis of fatty liver was made on the basis of obesity, alcoholism, and parotid enlargement. Sialograms were normal on both sides. A biopsy was taken from the right parotid (see Fig. 3-b). There was marked fatty infiltration separating a few small clumps of normal appearing acini. The ducts were normal.

Case 4. D.M.C., 664138. This 50-year-old white man was undergoing resection and plastic repair for an extensive basal cell carcinoma of the

Fig. 3-a. Case 3. Bilateral parotid enlargement in an obese woman.

left side of his face. He was moderately obese and had consumed two bottles of beer daily and a few highballs on week ends, but had abstained since the development of homologous serum hepatitis two years prior to biopsy. Bilateral parotid hypertrophy was noted as an incidental finding. A biopsy of the right parotid gland (see Fig. 4) was done while a free full thickness skin graft was being taken from behind the ear. The section contained about 50 per cent acini and 50 per cent fat. The acinar cells are packed tightly with zymogen granules as shown stained with periodic acid Schiff.

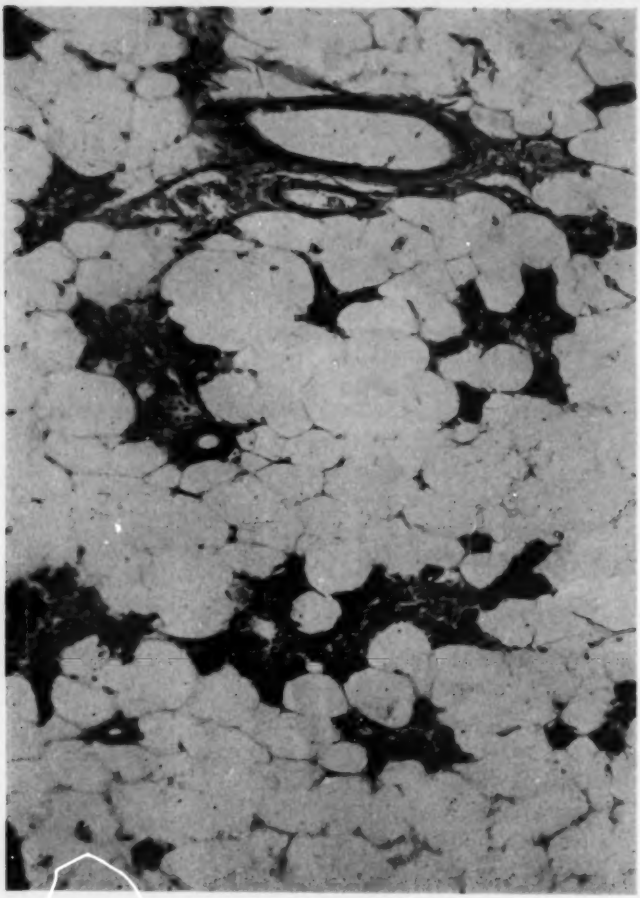

Fig. 3-b. Case 4. Marked replacement of parotid acini by fat cells.

Case 5. M.D., 470417. This somewhat obese 58-year-old white woman presented with a "walnut sized" mass in the right parotid area for two months. Biopsy of the mass (see Fig. 5) shows marked fatty infiltration. The remaining secreting cells appear to be packed with zymogen granules.

Case 6. R.O., 475301. This patient was a 37-year-old white male who had a diffuse asymptomatic swelling of the right parotid gland for two months. He was not obese and drank alcohol very rarely. A sialogram

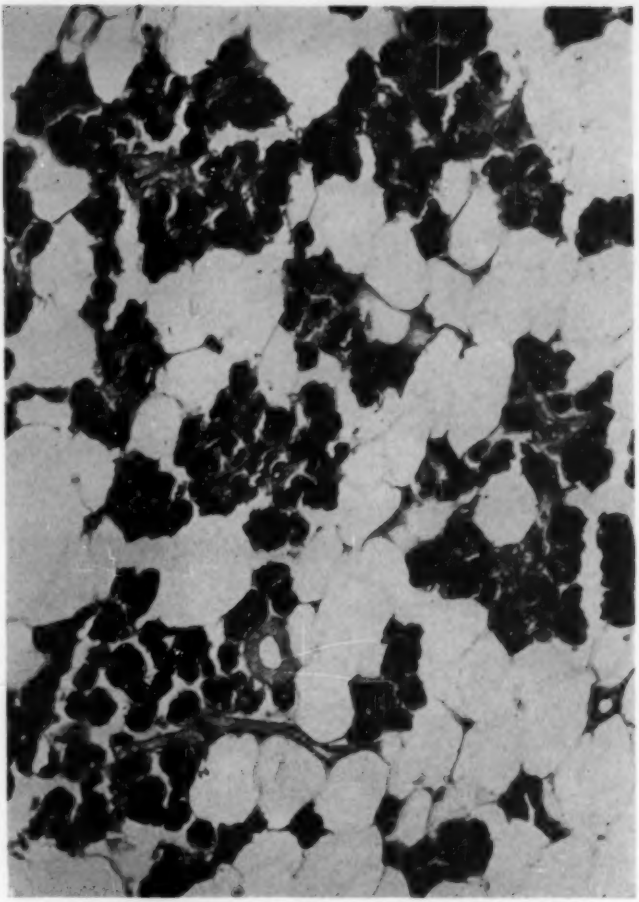

Fig. 4. Case 4. Parotid biopsy stained with periotic acid Schiff showing cells packed with zymogen granules.

was normal (see Fig. 6-a). Biopsy (see Fig. 6-b) showed mainly hypertrophy of the acinar cells with about the normal percentage of fat cells.

Case 7. M.F., a 59-year-old white woman, was seen with a diffuse enlargement of the right parotid gland which was clinically double the normal size for about four months. A biopsy was performed which showed hypertrophy of the secreting cells, increased zymogen granules, and an approximately normal quantity of fat.

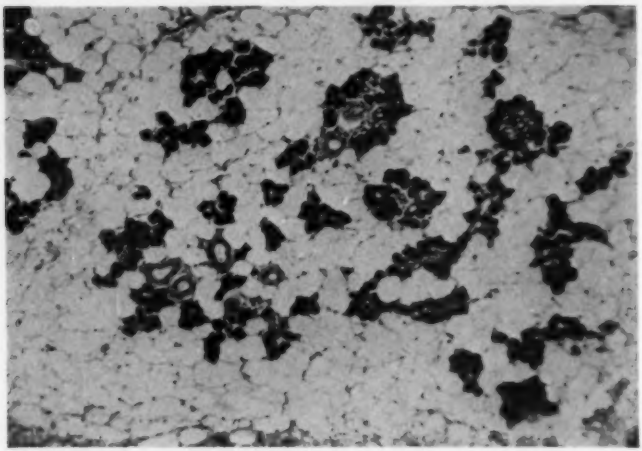

Fig. 5. Case 5. Considerable infiltration and increase in zymogen granules in a unilateral "walnut size" parotid mass.

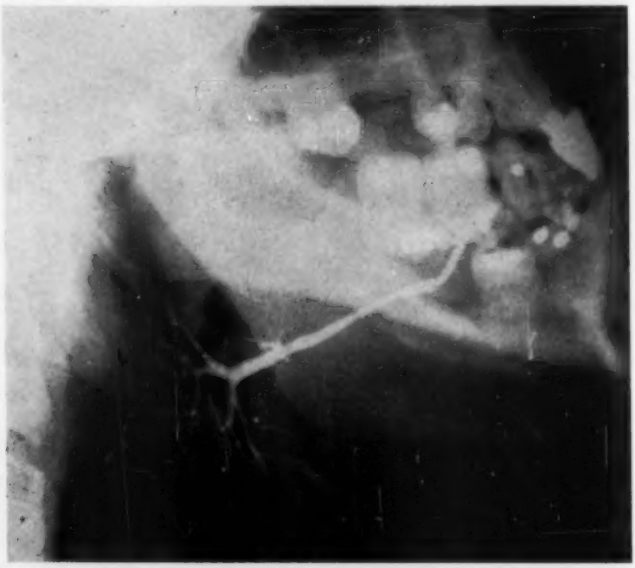

Fig. 6-a. Case 6. Normal parotid sialogram.

DISCUSSION.

Some of the literature has been reviewed on chronic enlargement of the parotid glands which are neither neoplastic nor inflammatory. Seven such cases have been described from our material. They fall roughly into three groups:

1. Ideopathic hypertrophy (two cases), both bilateral.

2. Fatty infiltration (two cases), one bilateral and one unilateral.

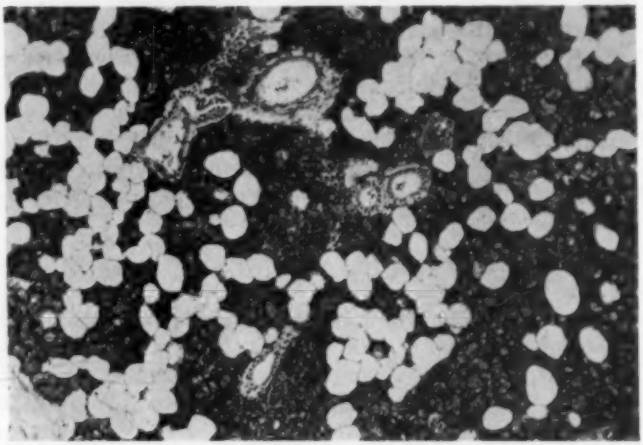

Fig. 6-b. Case 6. Parotid biopsy. Hypertrophy of acini in a unilateral enlargement.

3. A combination of one and two-three cases—one bilateral and two unilateral.

Case 1 would probably fit into the hypertrophy described in the literature associated with a generalized dietary deficiency; however, Case 2 had no obvious nutritional abnormality. Both these patients were psychoneurotic.

Cases 3 and 4 correspond to the bilateral hypertrophy seen in obese alcoholics with or without definite evidence of cirrhosis.

Case 4 is similar to that reported by Gilman, Schwartz and Gilman.

Cases 6 and 7 reveal similar pathological findings. A mixture of hypertrophy and fatty infiltration was found with acini and fat cells in roughly equal proportions. The acini were measured and found to be definitely larger than normal. The involvement was unilateral.

CAUSE OF THE ENLARGEMENT.

Apparently two factors may be responsible for the clinical enlargement of the glands in these patients. The first is an increase in the size of the individual acini. This is found mainly in glands which show a marked predominance of acini over fat cells. In Case 1, measurement of the acini showed that they were almost double the normal size, while in Case 6 and Case 7 the acini were about one-third oversize. There is no evidence for an increase in number of acinar cells.

Secondly, there may be an increase in the number of fat cells. In cases 3 and 5, which showed many more fat cells than acini, the acini were only slightly enlarged or within normal limits. The fat cells were measured and found to be of similar size to those found in normal parotid glands. The reason for the accumulation of fat is obscure. There is, of course, a widely variable number of fat cells in the normal parotid gland, and fat cells replace the acini which atrophy in Sjogren's disease and which are destroyed by X-ray therapy. In several of our cases an increase in number of fat cells must have contributed to the enlargement. Obviously in intermediate forms the clinical swelling is due to a combination of acinar hypertrophy and fatty infiltration.

Our material would seem to support Sandstead's and coworkers' thesis that the hypertrophy and fatty infiltration are different stages of the same process; however, etiology and pathogenesis remain obscure. Dietary deficiency undoubtedly plays a role in some cases, but most of our patients had no obvious nutritional defect. The occurrence of unilateral involvement in three of our cases would seem to

indicate that local factors may also be implicated. Two of our patients were psychoneurotic, but the remainder appeared reasonably stable. The striking occurrence of an increased number of zymogen granules in the cells and the relative absence of resting acini cannot be explained although it would appear the gland was responding to excessive prolonged stimulation.

Parotid swellings, due to idiopathic hypertrophy and fatty infiltration, are important only that they should be recognized as absolutely benign entities and not confused with inflammatory or neoplastic parotid disease. The bilateral enlargements in malnutrition and obese alcoholics should present no diagnostic problem. No local treatment to the parotid is indicated. The unilateral diffuse cases and those presenting as localized masses present clinical problems as these lesions often cannot be differentiated from certain inflammatory or neoplastic lesions. A sialogram should be done which will be normal in hypertrophy-fatty infiltration. Localized lesions should be removed, and diffuse lesions should be biopsied. If the patient wished treatment for a cosmetic reason, a partial parotid resection could be performed.

SUMMARY.

1. The literature has been reviewed on parotid enlargements in nutritional disturbances.

2. Seven cases of parotid swelling are presented, in which the pathological changes show a striking absence of inflammatory reaction or neoplastic change. Changes seen are quantitative rather than qualitative. Increase in size of the individual cells and a striking increase in number of zymogen granules are seen. The amount of fat varies from practically none to over 90 per cent in different cases. The ducts are normal and sialograms done in four cases showed no abnormality.

3. Parotid biopsy is an innocuous procedure and should be done frequently for diagnosis for accumulation of more pathological information about these conditions.

4. Etiology and pathogenesis are obscure. The hypertrophy

and fatty infiltration are probably manifestations of the same process. Enlargement is either due to an enlargement of the acini or to an increased number of fat cells or a combination of both. Increased number of zymogen granules and lack of resting acini are regularly seen. The condition is usually called ideopathic hypertrophy of the parotid if acinar swelling predominates, or fatty infiltration if fat cells predominate.

5. The submaxillary glands may show the same reaction either alone or accompanying the parotid enlargement.

6. Nutritional factors are involved in some cases, but are absent in others.

7. The condition is quite innocuous, and its only importance is that it might be confused with more serious parotid lesions.

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ANNOUNCEMENT.

The Annual Spring Meeting of the Alumni Association of the New York Eye and Ear Infirmary will take place April 4-7, 1960.

The meeting will be oriented towards pediatric otolaryngology. Symposia will be offered on: Nystagmus as a Pediatric Problem; Diagnosis and Rehabilitation of Hearing Problems in Children; and Ankylosis of the Stapes. There will also be a closed circuit television demonstration of surgical procedures. In addition, courses will be offered in: Allergy and Immunology in Children; X-ray Diagnosis in Ear and Sinus Problems in Children; Pediatric Laryngeal Problems, and Secretory Otitis Media in Children.

Additional information may be obtained by writing to Dr. John R. Finlay, Secretary, Alumni Association, 218 Second avenue, New York 3, N. Y.

OSTEOMA OF THE EUSTACHIAN TUBE.*†

VICTOR H. HILDYARD, M.D.,
and
W. E. COMPERE, JR., M.D.,

Los Angeles, Calif.

Surgery for the chronically infected ear is now performed to improve the hearing as well as to eradicate disease. Before one performs such surgery it is necessary to evaluate thoroughly the function of the middle ear mechanism and the Eustachian tube. To study the function of the Eustachian tube a tympanograph is made as first suggested by House and developed by Compere. This procedure consists of placing radiopaque contrast media in the middle ear and determining the time required for it to be emptied through the Eustachian tube.

It was while employing tympanography that an osteoma was discovered at the tympanic orifice of the Eustachian tube. A review of the available literature does not mention osteoma as a possible cause for obstruction of the Eustachian tube and, therefore, I am making this case report.

Bones developing in the membrane, which include most of the bones of the skull, ossify directly in a primitive connective tissue. The primitive connective tissue cells become changed into osteoblasts, which lay down bone. Such bones are not liable to the development of primary tumors. In general terms, it may be said that osteoblasts may give rise to osteomas, which are not true tumors, but rather a formation of new bone arising from the surface and known as exostosis. They are usually caused by irritation of the surface of the bone. In this case the chronic discharge could be the irritant.

*From the Department of Otolaryngology, University of Southern California School of Medicine.

†Sponsored by the Los Angeles Foundation of Otology.

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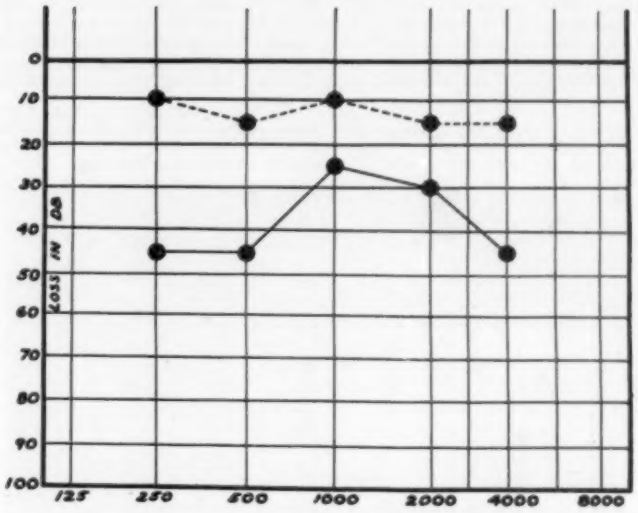

Fig. 1.

Fig. 2. Tympanogram revealing the osteoma obstructing the tympanic portion of the Eustachian tube.

CASE REPORT.

Miss M.R., age 22, was first examined on Nov. 29, 1957, because of a hearing loss since the age of seven years. The patient stated that she developed drainage from both ears at the age of seven years. The infection in the left ear subsided with therapy, but the right ear continued to drain intermittently throughout the years.

Examination of the right ear revealed a central perforation approximately 4 mm. in diameter. There was no drainage from the ear. Examination of the left ear revealed a healed central perforation. The remainder of the examination of the ears, nose and throat was essentially negative.

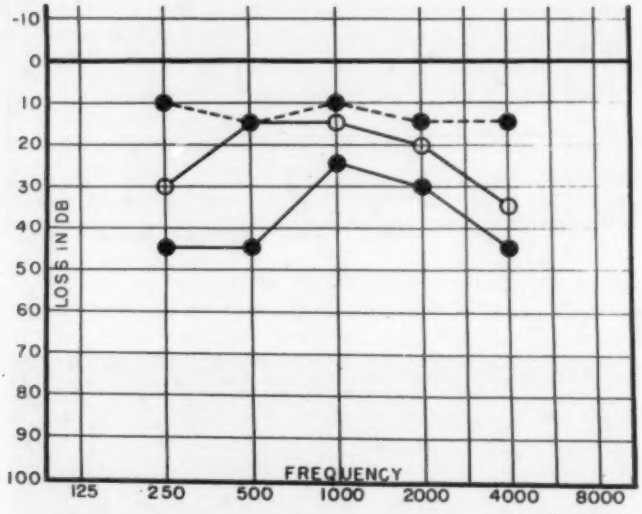

Fig. 3. Postoperative Audiogram.

Auto inflation of the right Eustachian tube was not effective. The acoustic probe revealed that sound was perceived better through the ossicular chain than through the promontory.

Audiometric examination revealed a conductive type hearing impairment of 33 db in the right and 23 db in the left ear, as indicated in the audiogram.

A tympanogram by Compere revealed obstruction to the contrast media at the tympanic orifice of the Eustachian tube with a clear outlining of a small osteoma.

A myringoplasty was performed on her right ear on Jan. 4, 1958. At the time of this procedure it was noted the tympanic portion of the Eustachian tube was completely obstructed by a smooth, hard, white,

rounded growth of bone from the inferior bony wall of the Eustcahian tube, which measured approximately 4 mm. in diameter. The growth was readily removed with a small curette and a skin graft was placed over the de-epithelized ear drum.

The patient made an uneventful recovery, and the hearing improved to a 17 db average level, as per the postoperative audiogram.

The postoperative X-ray of the right temporal bone on June 20, 1958, revealed a postoperative defect where the osteoma had been removed.

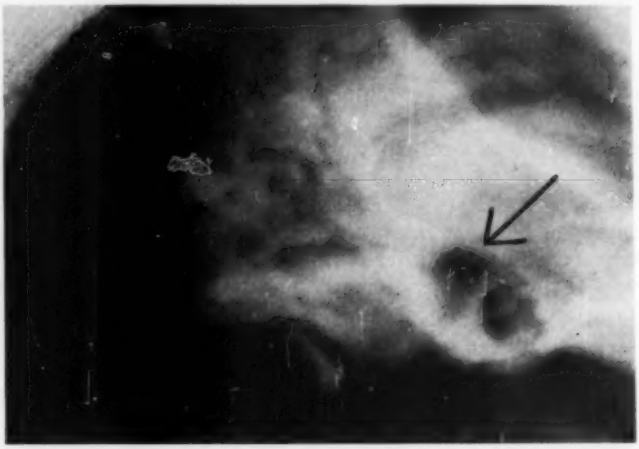

Fig. 4. Postoperative X-ray showing defect after removing osteoma.

SUMMARY.

An osteoma obstructing the tympanic portion of the Eustachian tube was diagnosed preoperatively by means of a tympanogram. This entity may now be added to the other causes of possible Eustachian tube obstruction.

SKELETAL MUSCLE RHABDOMYOMA OF THE LARYNX: REPORT OF A CASE.*

HOWARD W. SMITH, M.D.,

New Haven, Conn.

A benign tumor arising in skeletal muscle and composed chiefly of muscle fibers containing both longitudinal and cross striations is a striated muscle rhabdomyoma. Such a tumor is exceedingly rare, for Willis,[1] 1950, stated that benign rhabdomyomas of skeletal muscle are unknown in spite of the very large mass of voluntary muscle in the body. Rhabdomyomas of skeletal muscle are exceedingly rare. The classical description was recorded by Ewing,[2] in 1919. He described this tumor as "composed chiefly of a system of parallel bundles or intertwining strands of striped muscle fibers, supported by adult or embryonal connective tissue. The cells were usually thin, much elongated and rarely they were branching. Both longitudinal and cross striations may be pronounced, or the cross striations may be present in a part of the cell only or be entirely missing in embryonal or spindle shaped cells which still retained their longitudinal fibrillations. The cells may closely resemble smooth muscle cells in some areas."

A number of laryngeal tumors have been reported under the term "rhabdomyoma" but described as granular cell myoblastomas.[3,4] Imperatori,[3] 1933, reported a case of rhabdomyoma of the larynx. He described the tissue mass as coarsely papillary and firm with a cut surface which was homogenous and pale yellow in color. Microscopic sections were reported to show striated muscle fibers of varying size and shape, and a small amount of fibrous tissue intermingled with the muscle fibers. Parsons,[5] 1955, reported a case of rhabdomyoma of the sternothyroid muscle. He could find no

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previous case reports of rhabdomyoma arising from skeletal muscle.

A closer look at the photomicrographic illustrations of the case reported by Parsons, in 1955, raises certain objections to the use of the title rhabdomyoma, for the lesion as demonstrated was composed chiefly of granular cells having questionable striations and showing little, if any, resemblance to striated muscle.

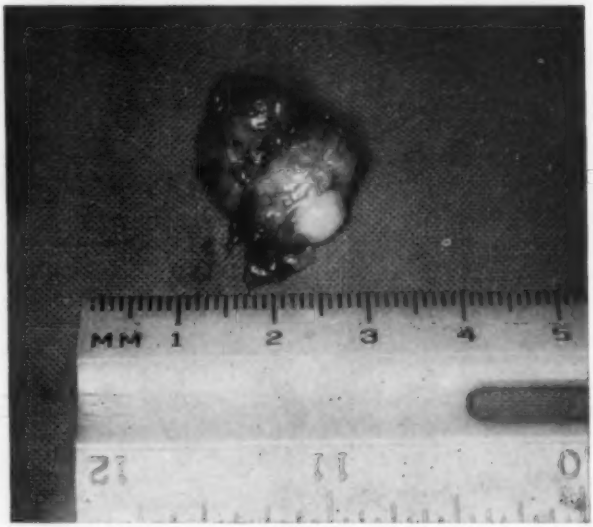

Fig. 1. Photograph of striated muscle rhabdomyoma removed from the larynx.

Seventeen cases of granular cell myoblastoma of the larynx have been reported.[6,7] It is possible that a number of this group may be true rhabdomyomas, as striated muscle elements have been noted in the microscopic sections in a few of the cases. Schirmer and Meyer[8] attributed such findings to erroneous interpretation of the relation between neoplastic elements and pre-existing striated muscle fibers. The relation of granular-cell myoblastoma to striated muscle seems to be

more tenuous as time goes by. The idea that the granular cells are embryonic precursors of striated muscle fibers is now generally rejected.[6] That it may be a degenerative lesion of muscle is supported by Willis,[9] 1953. Still others believe it to have a neurofibromatous nature.[10]

The only known case report of a benign striated muscle rhabdomyoma meeting Ewing's classical description, other than the case reported by Imperatori, is that of a rhabdomyo-

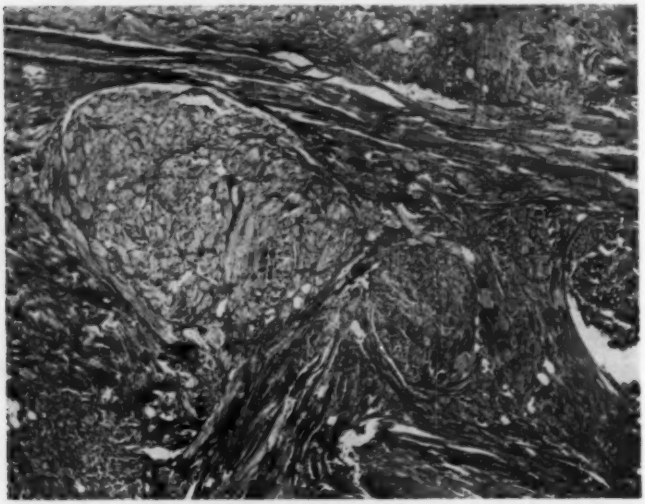

Fig. 2. Photomicrograph showing striated muscle fibers going in different directions.

ma of the nose reported by Reitter in 1921.[11] He described the tissue as consisting of irregular bundles of fibers closely resembling those of striated muscle. The bundles consisted of a varying number of fibers running in many directions without regular arrangement. Each fiber had a distinct sarcolemma and cross striations were visible. The fibers were found to be somewhat coarser than those of normal striated muscle. The bundles of fibers were separated by

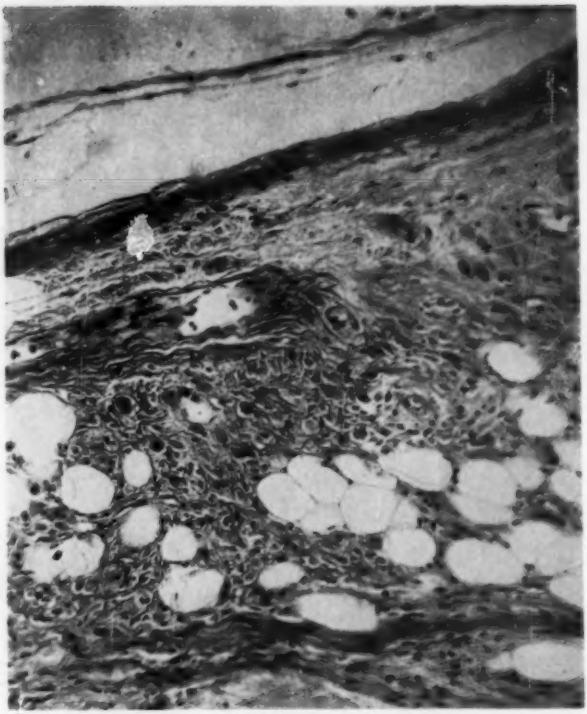

Fig. 3. Photomicrograph showing fatty infiltration.

areas of delicate connective tissue. No reference to granular cells was found in the histopathologic report.

CASE REPORT.

A 39-year-old white female was admitted to the hospital because of longstanding hoarseness, a mass in the larynx, and a recent episode of choking.

At age eight, the patient was examined because of hoarseness. An irregularity of the larynx was observed at that time, but no treatment was recommended. The hoarseness had remained unchanged since that time.

Three weeks prior to admission the patient had an upper respiratory infection and complained of choking and the presence of a mass in her throat. Examination revealed an irregularity of the larynx.

Admission physical examination showed a well developed and well

Fig. 4. Photomicrograph showing replacement of muscle fibers with dense collagenous tissue and absence of capsule.

nourished white female in no distress. The voice was very husky, and there was much wasting of the air stream causing her to speak slowly and in short sentences. Physical findings were essentially normal, except for those observed by mirror laryngoscopy. The right half of the glottis was occluded by a medial displacement of the right lateral wall. The right true vocal cord could not be seen. There was limited movement of the right false cord. The remainder of the larynx appeared normal. Food and liquids were swallowed without difficulty. No abnormal masses were palpable in the neck. There was slight tenderness over the right half of the larynx.

Admission blood and urine studies were normal. A metastatic X-ray series and barium swallow were normal. Special X-rays of the larynx revealed asymmetry of the tracheal air column on the right. This asymmetry appeared as an impingement of the airway at the level of the right glottic and subglottic area.

On the second hospital day a tracheotomy was performed under local anesthesia and followed by direct laryngoscopy under general anesthesia.

The whole right half of the intrinsic laryngeal structures appeared to be forced over to the midline. The right true vocal cord appeared to be displaced medially and downward. There was a firm moveable mass between the true and false vocal cords which could be demonstrated by displacing the overlying mucosa and intrinsic muscles. Immediately following laryngoscopy, a laryngofissure was performed. A soft moveable mass was palpated in the right half of the larynx. It appeared to lie against the laryngeal cartilage deep to the mucosa and intrinsic muscles. The major portion appeared to be between the true and false cords, but there was definite extension beneath the true cord into the subglottic area. The right vocal cord was detached anteriorly and retracted medially

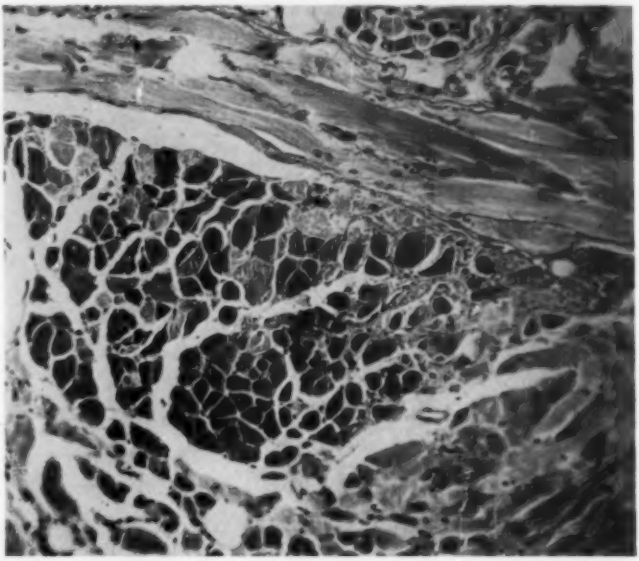

Fig. 5. Photomicrograph of striated muscle fibers in cross section. Note peripheral position of nuclei.

to expose the tumor mass. The tumor was intimately related to the intrinsic musculature and lay between it and the perichondrial lining of the right alar cartilage. The tumor was quite soft and polypoid. No muscle fibers appeared to run into the mass. It seemed to be intimately related to the muscles of the larynx but in no way attached to them. The mass was yellow in color and easily distinguished from the muscle fibers. When the mass was removed the true cord was re-attached in its normal position and the larynx closed. There were no complications following surgery and the tracheotomy tube was removed on the eighth postoperative day. Gradual improvement of the voice has been observed since that time.

The specimen sent to the pathology laboratory was reddish-yellow, soft,

lobulated mass measuring 3x2x1 cm. (see Fig. 1). The cut section appeared to have a lobulated architecture with small yellow pock-marks. Microscopic examination revealed a mass of striated muscle fibers haphazardly arranged in bands and large bundles going in different directions (see Fig. 2). There was also a moderately large amount of fibro-adipose tissue and several areas showed striated muscle infiltrated with fatty elements (see Fig. 3). There was some hyalinazation of muscle fibers and replacement with dense collagenous tissue in a few areas (see Fig. 4). Examination of the muscle fibers under high power in cross section (see Fig. 5), and in longitudinal section (see Fig. 6), revealed peripheral placement of nuclei and unequivocal longitudinal and cross

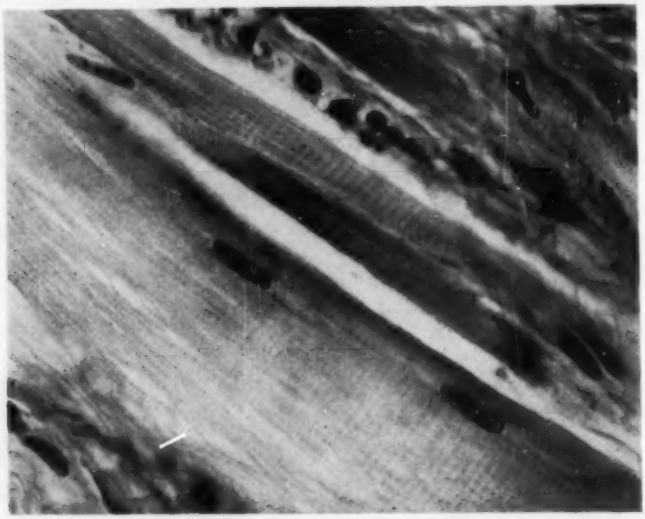

Fig. 6. Photomicrograph of striated muscle fibers in longitudinal section. Note presence of both longitudinal and cross striations.

striations. The nuclei of the muscle fibers did not seem unusual in size, staining characteristics, or configuration, and there was no indication of malignant transformation.

DISCUSSION.

The above case report conforms to the classical description of benign rhabdomyoma of skeletal muscle as described by Ewing in 1919. The only known case reports which bore any true resemblance to Ewing's description was a rhabdomyoma of the larynx reported by Imperatori,[3] 1933, and a rhabdomyoma of the nose reported by Reitter[10] in 1921.

The tissue mass in the present case report bears little, if any, histologic resemblance to the lesions described in the cases of myoblastoma tabulated by MacNaughtan, et al.,[6] 1954, and Hinton, et al.,[7] 1958. Their lesions were for the most part superficial projections from the true or false cords or raised plaques intimately associated with the epithelium. Their removal in most instances was accomplished by peroral laryngoscopy and forceps removal. When one understands the procedure it is easy to interpret a report on the histopathology which reads "striated muscle fibers were noted." In addition, MacNaughtan considered the granular cells myoblastoma to consist of sheets or clumps of large round cells, the cytoplasm of which is always granular and often acidophilic. The present case fails to qualify for definition as granular cell myoblastoma.

Now that the origin of the granular cell has been challenged as to its being the precursor of skeletal muscle and even considered as a degenerative process of muscle or of neurogenic origin, one can no longer consider a tumor arising in striped muscle, made up primarily of varying arrangements of striated muscle elements as a granular-cell myoblastoma. The case reported by Parsons, et al.[4] would be somewhat more acceptable as a benign striated muscle rhabdomyoma if the striations had been demonstrated on muscle fibers where proper and unequivocal distinction could be made, instead of showing waves in the granules of large round cells presumably cut on cross section.

The obscure and broad origin of granular cell tumors and their frequent classification as benign rhabdomyomas of skeletal muscle should not be so gross as to include those tumors arising in skeletal muscle showing true muscle fibers, unequivocal longitudinal and cross striations and containing no granular cells.

SUMMARY.

A true benign striated muscle rhabdomyoma of the larynx presenting symptoms for over 30 years has been reported. This tumor conforms to the classical description of the striated muscle lesion presented by Ewing in 1919, except for the fibro-

adipose elements which may also qualify the tumor as a hamartoma. One previous case report of a striated muscle rhabdomyoma occurring in the larynx was found.

The continued interchangeable use of the terms "granular cell myoblastoma" and "rhabdomyoma" to describe tumors composed of large vacuolated granular cell masses of dubious origin is deplored.

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442 Temple St.

THE UNIVERSITY OF MICHIGAN MEDICAL CENTER.

The annual Otolaryngology Conference at The University of Michigan will be held April 21, 22, and 23, 1960, under the direction of Dr. James H. Maxwell, Chairman of the Department of Otolaryngology.

Applications may be addressed to the Department of Postgraduate Medicine, University Hospital, Ann Abor, Mich.

A METHOD OF REMOVING A BEAD FROM A BRONCHUS.

An Improvement in Procedure.

J. J. CANTOR,

Los Angeles, Calif.

During 1922, while in the employ of Dr. Simon Jesberg, I was called upon to design and construct an instrument for the removal of an oval bead with a hole 1/32-inch in diameter from the right bronchus of a 14-months-old child.[1]

The instrument that was devised was essentially a long crochet needle-like stylet of small enough cross section to pass easily through the hole in the bead. An eye in one end of the stylet was slotted so that it could be threaded (see Fig. 1). A continuous rubber band was hooked into this opening and then drawn down to a very thin cross section. The rubber band was cut from a sheet of the thinnest grade of dental rubber by two thin Gillette safety razor blades, which were clamped on opposite sides of a 3/32-inch piece of flat metal (see Fig. 2). The ends of the rubber strip were tied together to make a continuous rubber band.

In use, the proximal end of the stylet, with its tightly stretched rubber band, was passed into the hole in the bead (see Fig. 3) and the distal end of the rubber band, to which a thread had been attached in order that it not be lost down the bronchoscope, was gradually released. As the rubber band slowly contracted it increased in cross section, filled the hole in the bead and locked the bead to the stylet. The stylet, with its attached bead, was then withdrawn.

The success of the procedure depended upon the smoothness of the razor cuts, for it was found that even a small nick in the edge of the band would cause it to break when stretched. In this particular case, out of the hundreds of bands cut, only a few satisfactory bands were obtained. In the years fol-

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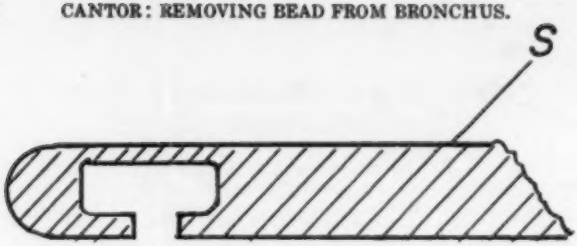

Fig. 1. Stylet.

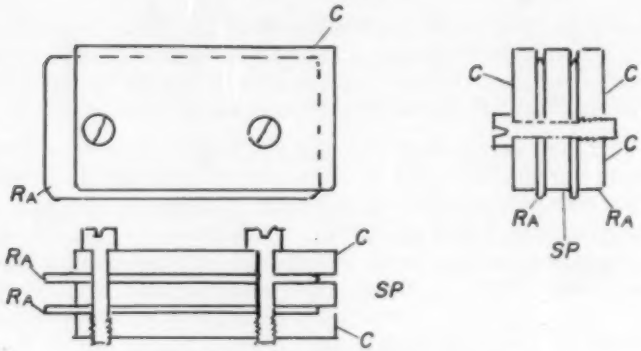

Fig. 2. Cutting Instrument (three views).

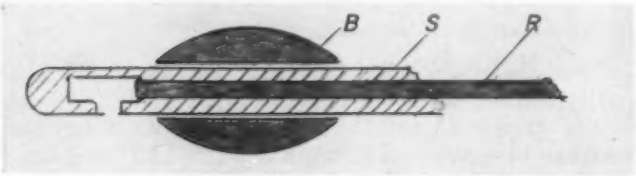

Fig. 3. Stylet in bead with rubber band stretched.

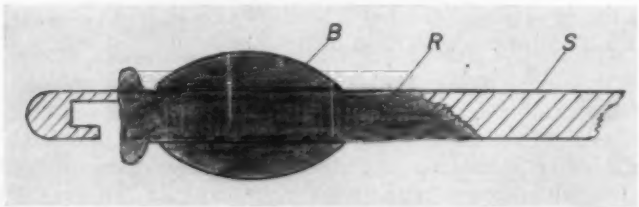

Fig. 4. Stylet, rubber band, and bead locked together.

Abbreviations: B—bead; C—clamp; R—rubber band; Ra—razor blade; S—stylet; Sp—spacer.

lowing the successful removal of this bead, I speculated upon numerous more efficient methods of producing smooth edged rubber bands.

Some 22 years later I was called in by Dr. Howard P. House, who had a similar bead case. At this time I employed a different method of cutting the rubber band.

First, I glued a piece of dental rubber between two sheets of kraft paper, using water soluble LePage's glue. The whole was allowed to dry upon a flat surface over night. When the sandwiched sheets of paper and rubber were thoroughly dry, I had no difficulty cutting smooth bands with the razor blade device shown in Fig. 2. The bands, with their attached paper, were dropped into a bowl of cold water. The water dissolved the glue, and the paper floated away from the rubber strip.

Dr. Howard P. House, assisted by Dr. Harold Owens, used the instrument described above to remove the bead through the tracheotomy wound.

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POSTGRADUATE COURSE.

The next postgraduate course in Laryngology and Bronchoesophagology to be given by the University of Illinois College of Medicine is scheduled for the period April 4 to 16, 1960. The course is under the direction of Dr. Paul H. Holinger.

Interested registrants will please write directly to the Department of Otolaryngology, University of Illinois College of Medicine, 1853 West Polk Street, Chicago 12, Ill.

SCIENTIFIC PROGRAM OF THE AMERICAN OTOLOGICAL SOCIETY, INC.

The Deauville Hotel, Miami Beach, Fla., March 13-14, 1960.

Early Embryology of the Auditory Ossicles and Associated Structures in Relation to Certain Anomalies Observed Clinically.

Barry J. Anson, Ph.D. (Med. Sc.);
Jerome S. Hanson, M.S. (by invitation), and
Shatik F. Richany, Ph.D. (by invitation).

Tissue Reaction Following Reconstruction of the Oval Window in Experimental Animals.

Richard J. Bellucci, M.D., and
Dorothy Wolff, Ph.D., (by invitation).

Human Cochlear Responses to Sound Stimuli.

John L. Bordley, M.D.;
Robert J. Ruben, A.B. (by invitation), and
George R. Nager, M.D. (by invitation).

Some Physiological Factors in Inner Ear Deafness.

Merle Lawrence, Ph.D.

Distribution of Hearing Loss in Various Population Centers.

Aram Glorig, M.D.

The Management of Middle Ear Lesions Simulating Otosclerosis.

Francis A. Sooy, M.D. (by invitation).

Experiences with the Stapedectomy Technique of Shea for the Surgical Treatment of Otosclerosis.

David Myers, M.D.;
Woodrow D. Schlosser, M.D. (by invitation), and
Richard A. Winchester, Ph.D. (by invitation).

Vein Plug Stapedoplasty.

Clair M. Kos, M.D.

Partial Stapedectomy.

J. V. Hough, M.D. (by invitation).

Experimental and Clinical Experiences in Oval Window Surgery.

Harold Schuknecht, M.D.

(Open discussion of last four papers.)

Acceleration as a Means of Determining the Sensitivity of the Components of the Non-Auditory Membranous Labyrinth.

Walter H. Johnson, Ph.D (by invitation);
J. Brydon Smith, M.D., and
Joseph A. Sullivan, M.D.

Localization of Monaurally Impaired Listeners.

Richard Viehweg, M.A. (by invitation).

Deafness Due to Mumps; Inner Ear Pathology.

John R. Lindsay, M.D., and
Patricia R. Davey, FRCS (Ed.) (by invitation).

Action of Enzymes on Effusions Obtained from the Middle Ear.

Carl F. Gessert, Ph.D. (by invitation);
Elizabeth S. Baumann (by invitation), and
Ben Senturia, M.D.

Tumors of the Temporal Bone in Their Reciprocal Behavior.

George Kelemen, M.D.

Labyrinthectomy.

Terence Cawthorne, FRCS.

The Lurking Latent Cholesteatoma.

Victor Goodhill, M.D.

DIRECTORY OF OTOLARYNGOLOGIC SOCIETIES.

(Secretaries of the various societies are requested to keep this information up to date).

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

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Meeting: Palmer House, Chicago, Ill., October, 1960.

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Meeting: Palace Hotel, Denver, Colo., May 12-14, 1960.

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Meeting: Chicago, Ill., October, 1960.

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Meeting: Deauville Hotel, Miami Beach, Fla., March 15-16, 1960 (Afternoons only).

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Meeting: Deauville Hotel, Miami Beach, Fla., March 18-19, 1960.

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Annual Meeting: October 1, 1960, Belmont Hotel, Chicago, Ill.

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Meeting: Jasper Park Lodge, Jasper National Park, Alberta, June 10-12, 1960.

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Meeting:

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Meeting:

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Secretary of Ophthalmology Section: Dr. Philip D. Shanedling.
Chairman of Otolaryngology Section: Dr. Robert W. Godwin.
Secretary of Otolaryngology Section: Dr. Francis O'N. Morris.
Place: Los Angeles County Medical Association Bldg., 1925 Wilshire Blvd., Los Angeles, Calif.
Time: 6:30 P.M. last Monday of each month from September to June, inclusive—Otolaryngology Section. 6:30, first Thursday of each month from September to June, inclusive—Ophthalmology Section.

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Meeting:

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Meeting:

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Meeting:

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Meeting: Seventh Pan American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology.
Time and Place: Miami, Fla., March, 1960.

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Meeting: University of Vienna, August 29 to September 1, 1960.

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Vice-Chairman: Dr. Bernard J. McMahon, Director of the Department and Clinical Professor of Otolaryngology, St. Louis University School of Medicine, 8230 Forsythe Blvd., Clayton 24, Mo.
Secretary: Dr. Albert C. Esposito, First Huntington National Bank Building, Huntington, W. Va. Formerly instructor of Ophthalmology, Ohio State University College of Medicine, Columbus, O.
Meeting: St. Louis, Mo., October 31, 1960, to November 3, 1960.

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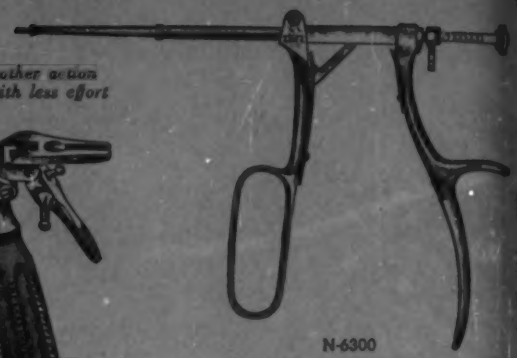

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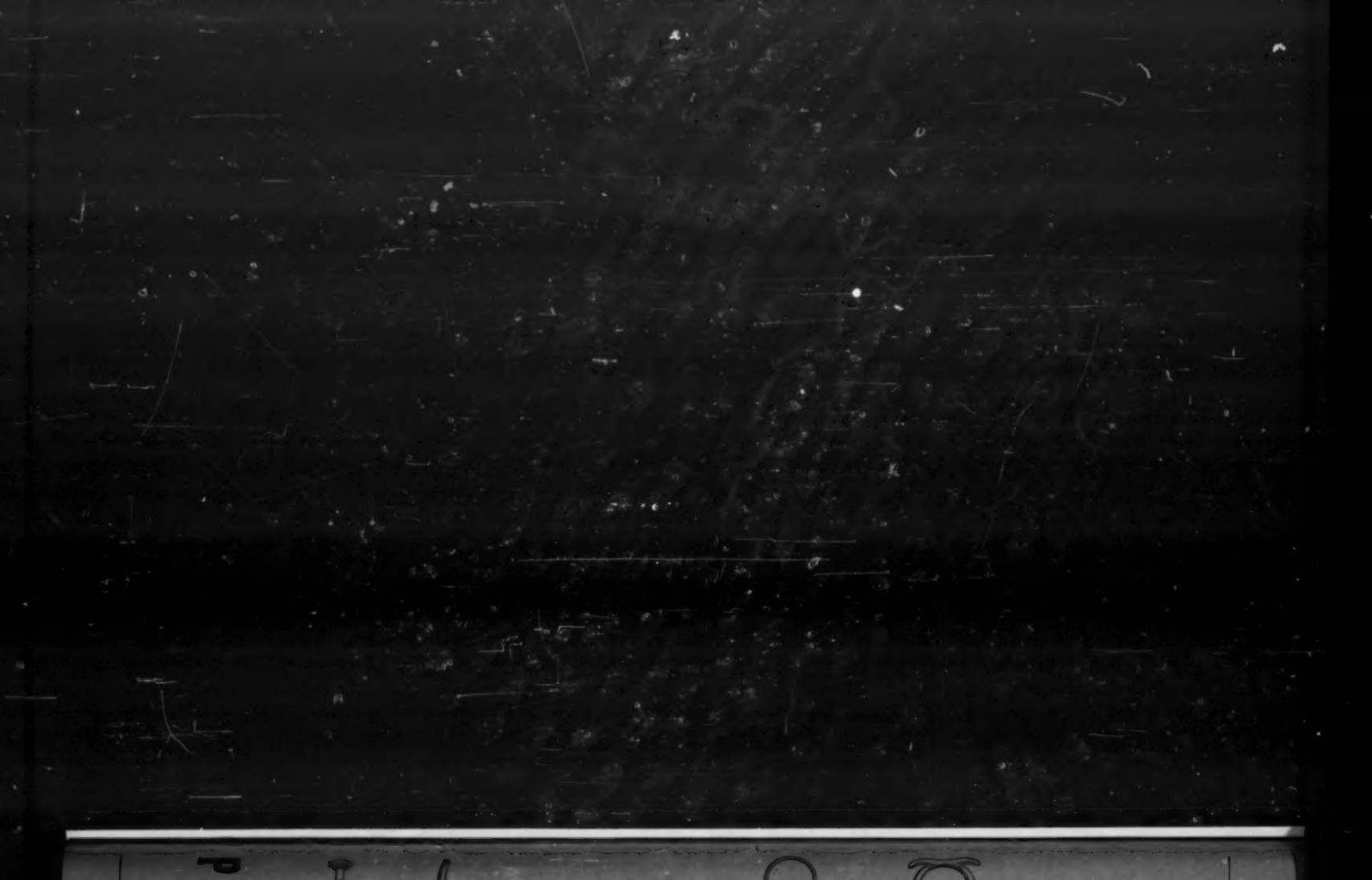

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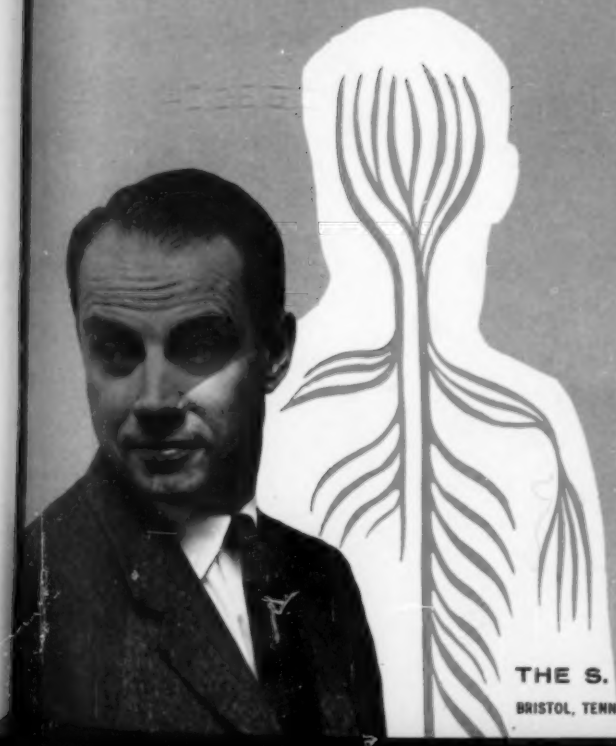

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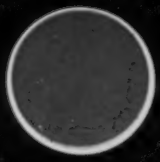

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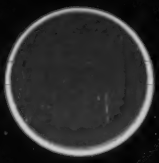

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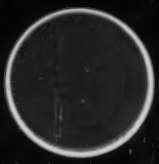

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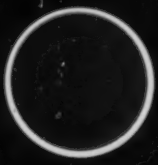

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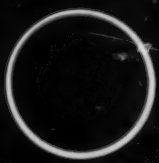

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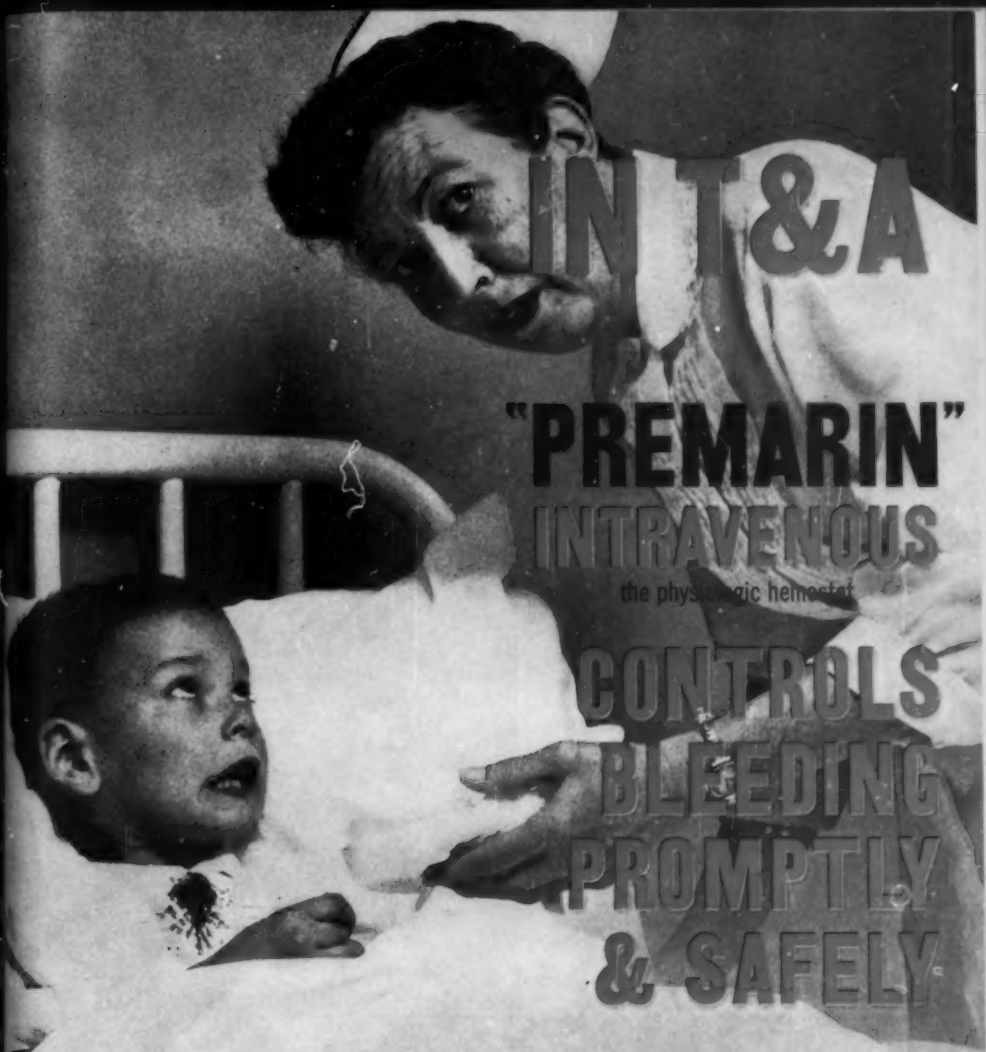

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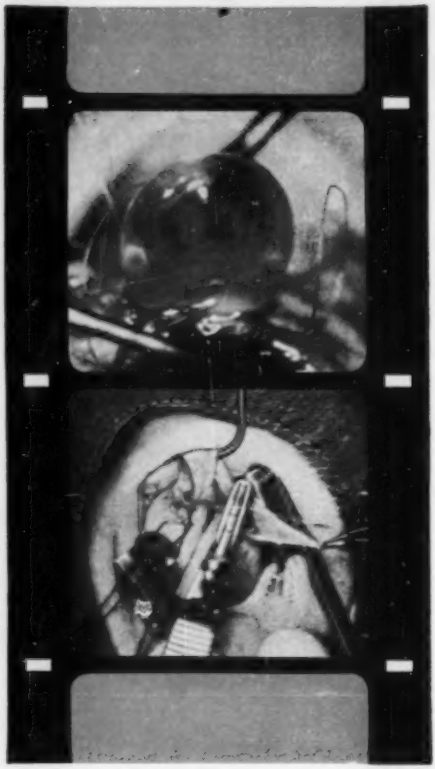

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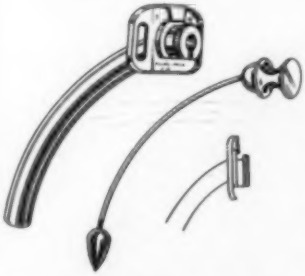

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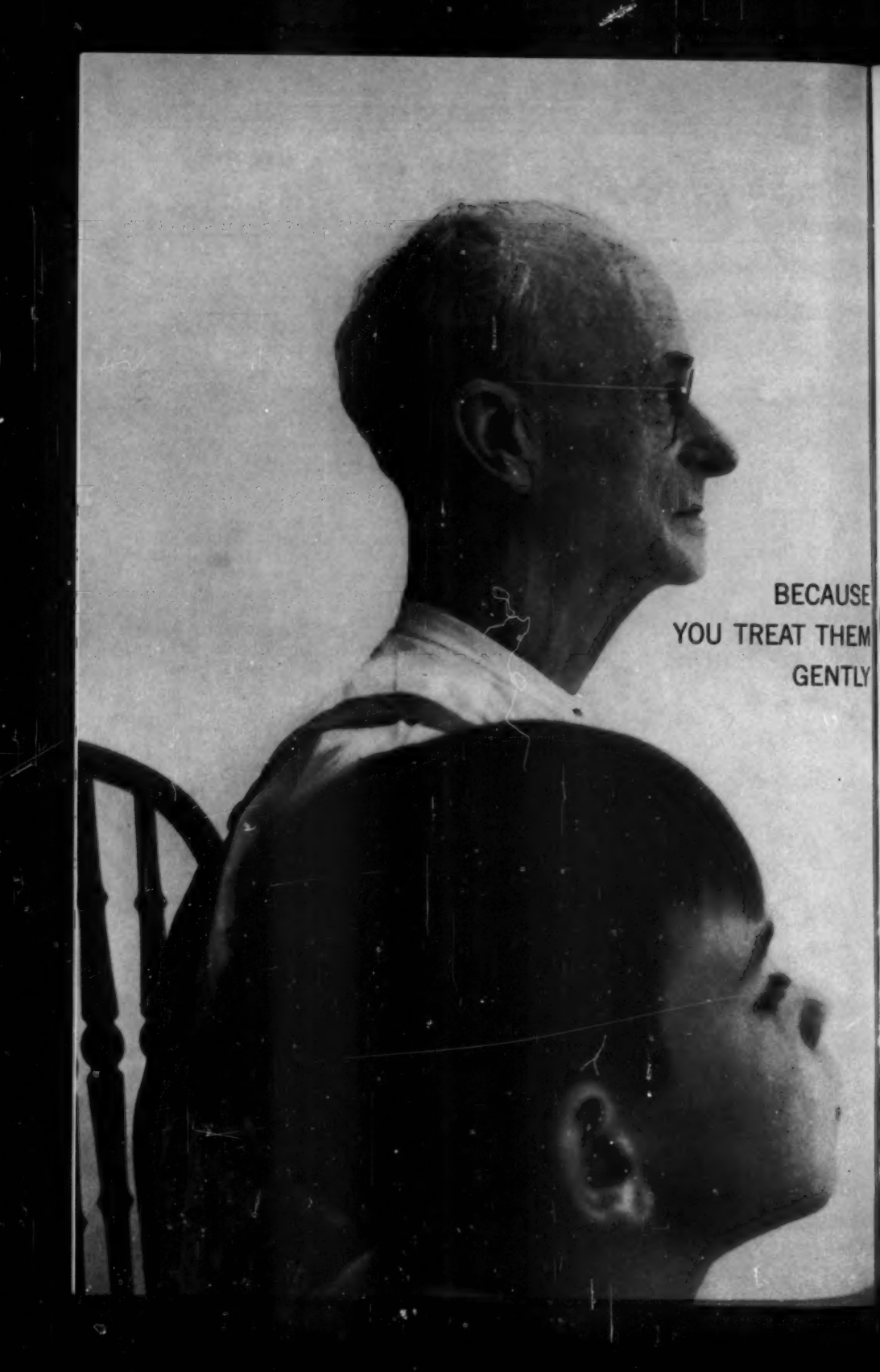
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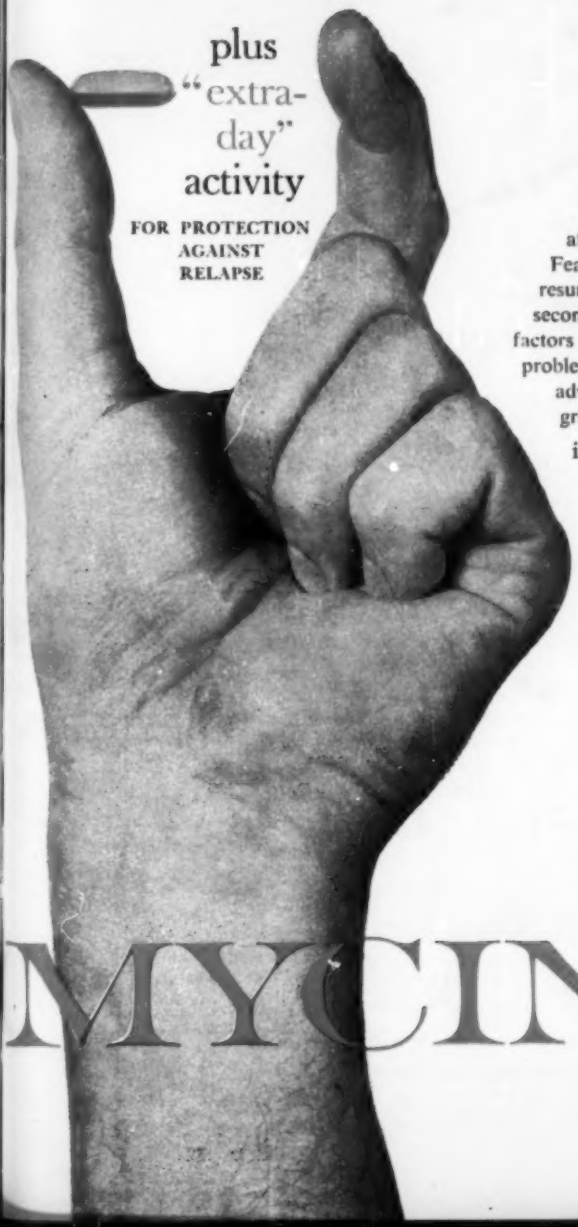

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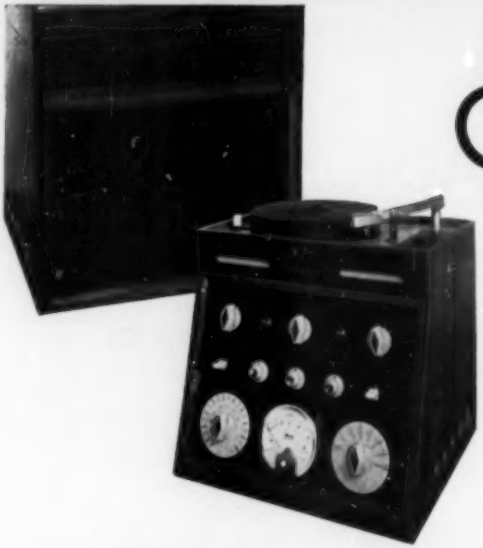

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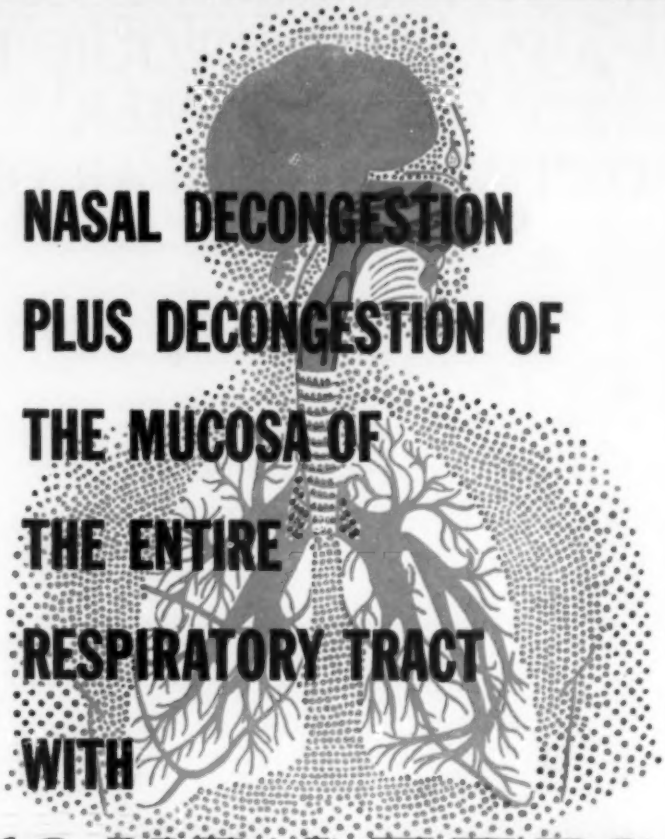

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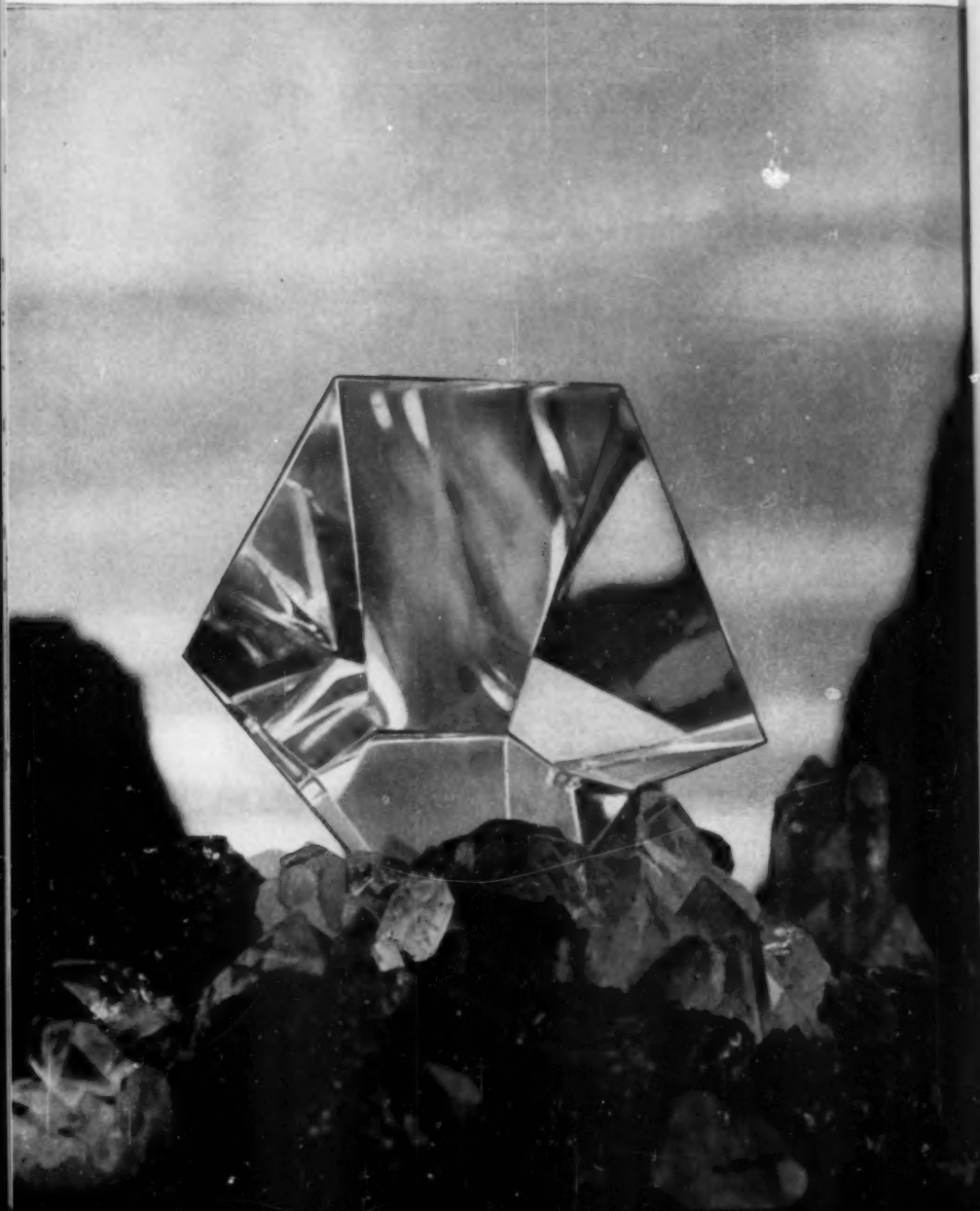

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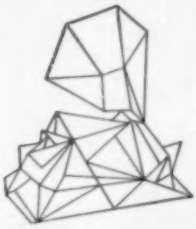

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2 mg. Tablet	q.i.d.
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References: (1) Gould, A. H. and Long, D. L.: Clinical Pharmacology and Therapeutic Use of Dexbrompheniramine Maleate (Disomer), a new Histamine Antagonist (submitted for publication). (2) Medical Department, White Laboratories, Inc.

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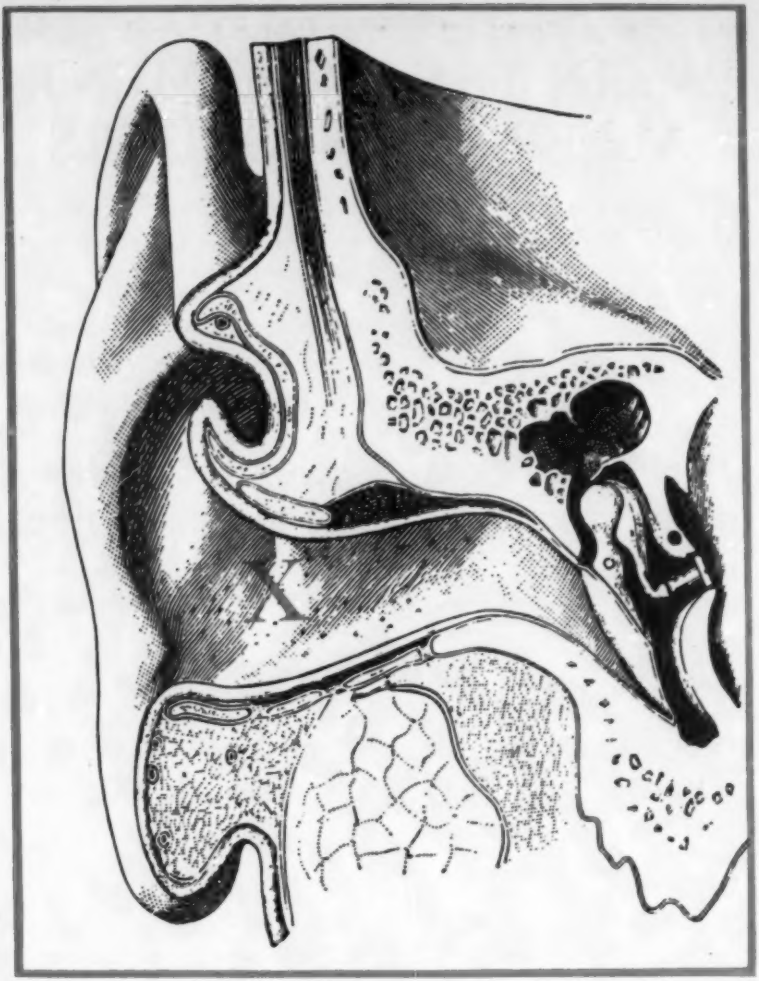

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*Lawson, G. W.: Diffuse Otitis Externa and Its Effective Treatment, Postgrad. Med. 22:501, (Nov.) 1957.

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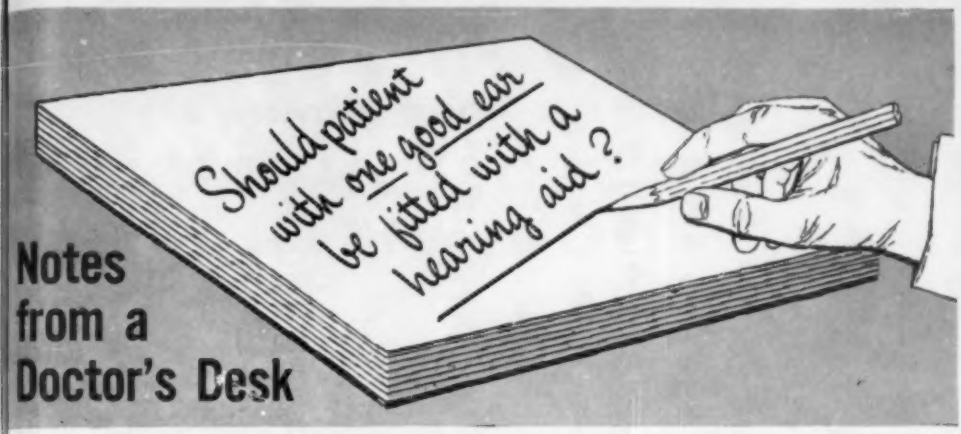

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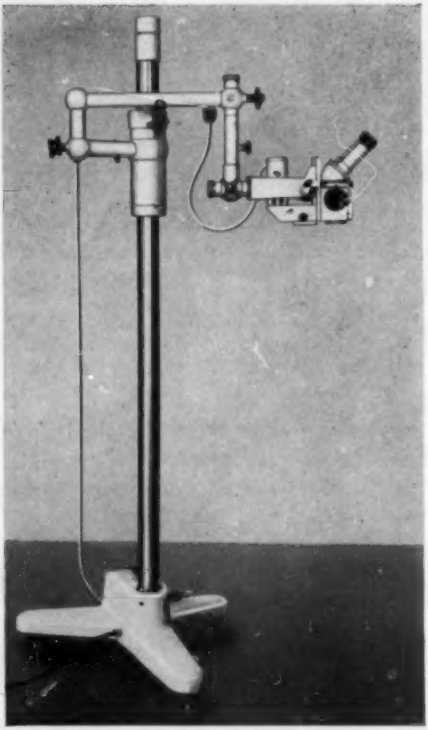

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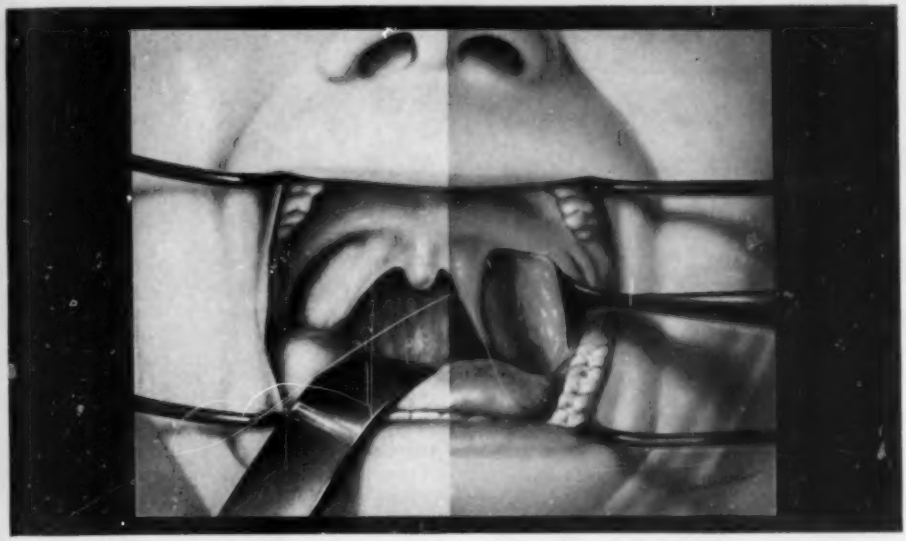

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**Granberry, C., and Beatrous, W.P.: The Effect of an Antibiotic Chewing Troche on Post-Tonsillectomy Morbidity, E.E.N.T. Monthly (May) 1957.*

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References: 1. Lhotka, F. M.: Illinois M. J. 112: 259 (Dec.) 1957. 2. Fabricant, N. D.: E.E.N.T. Monthly 37:460 (July) 1958. 3. Farmer, D. F.: Clin. Med. 5:1183 (Sept.) 1958.

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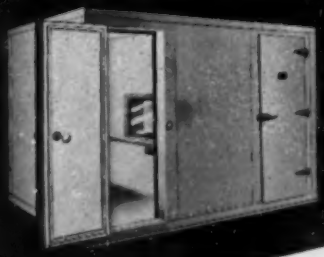

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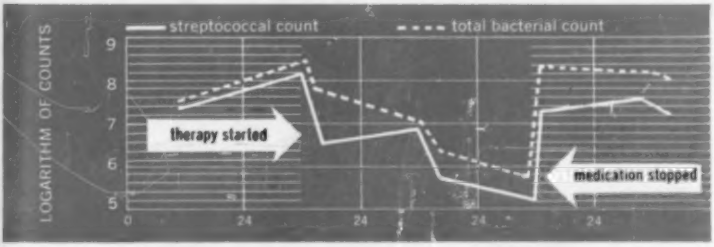

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